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TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY OF LONDON.

VOLUME THE THIRTY-NINTH.

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE SESSION 1887-88.

LONDON:
SMITH, ELDER & CO., 15, WATERLOO PLACE.
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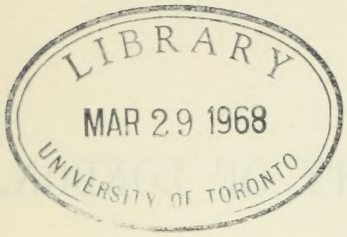

The present publication, being the Thirty-ninth Volume of Transactions, constitutes the Forty-second published Annual Report of the Pathological Society's Proceedings.

The Council think it right to repeat that the exhibitors are alone responsible for the descriptions given of the Specimens exhibited by them, the only change made in the Reports furnished by the authors being such verbal alterations as were absolutely necessary.

53, Berners Street, Oxford Street;
October, 1888.

7646

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Presidents of the Society.

ELECTED

1846 CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848 CHARLES ASTON KEY.
1850 PETER MERE LATHAM, M.D.
1852 CÆSAR H. HAWKINS, F.R.S.
1853 BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855 JAMES MONCRIEFF ARNOTT, F.R.S.
1857 Sir THOMAS WATSON, Bart., M.D., F.R.S.
1859 Sir WILLIAM FERGUSSON, Bart., F.R.S.
1861 JAMES COPLAND, M.D., F.R.S.
1863 Sir PRESCOTT G. HEWETT, Bart., F.R.S.
1865 THOMAS BEVILL PEACOCK, M.D.
1867 Sir JOHN SIMON, K.C.B., D.C.L., F.R.S.
1869 RICHARD QUAIN, M.D., F.R.S.
1871 JOHN HILTON, F.R.S.
1873 Sir WILLIAM JENNER, Bart., M.D., K.C.B., D.C.L., F.R.S.
1875 GEORGE D. POLLOCK.
1877 CHARLES MURCHISON, M.D., LL.D., F.R.S.
1879 JONATHAN HUTCHINSON, F.R.S.
1881 SAMUEL WILKS, M.D., F.R.S.
1883 JOHN WHITAKER HULKE, F.R.S.
1885 JOHN SYER BRISTOWE, M.D., F.R.S.
1887 Sir JAMES PAGET, Bart., D.C.L., LL.D., F.R.S.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, JANUARY 3RD, 1888.

President.

SIR JAMES PAGET, BART., D.C.L., LL.D., F.R.S.

Vice-Presidents.

THOMAS HENRY GREEN, M.D.
J. HUGHLINGS JACKSON, M.D., F.R.S.
JOSEPH FRANK PAYNE, M.D.
RICHARD DOUGLAS POWELL, M.D.
WILLIAM MORRANT BAKER.
MARCUS BECK, M.S.
SIR JOSEPH LISTER, BART., D.C.L., LL.D., F.R.S.
HENRY MORRIS, M.B.

Treasurer.

WILLIAM CAYLEY, M.D.

Council.

CHARLES EDWARD BEEVOR, M.B.
HENRY RADCLIFFE CROCKER, M.D.
DAVID WHITE FINLAY, M.D.
JAMES KINGSTON FOWLER, M.D.
JAMES FREDERIC GOODHART, M.D.
WALTER BAUGH HADDEN, M.D.
EDWARD EMANUEL KLEIN, M.D., F.R.S.
STEPHEN MACKENZIE, M.D.
JOSEPH ARDERNE ORMEROD, M.D.
WILLIAM HALE WHITE, M.D.
WILLIAM ANDERSON.
ANTHONY ALFRED BOWLBY.
HENRY TRENTHAM BUTLIN.
VICTOR HORSLEY, B.S., F.R.S.
JOHN HAMMOND MORGAN.
BERNARD PITTS, M.B.
A. QUARRY SILCOCK, M.D.
JOHN BLAND SUTTON.
CHARTERS JAMES SYMONDS M.S.
FREDERICK TREVES.

Honorary Secretaries.

SIDNEY COUPLAND, M.D. | RICKMAN JOHN GODLEE, M.S.

Trustees.

RICHARD QUAIN, M.D., F.R.S. | GEORGE D. POLLOCK.
SAMUEL WILKS, M.D., F.R.S.

*** *Members are requested to indicate to the Secretaries corrections when necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna.
BRUECKE, ERNST, M.D., Professor of Physiology in the University of Vienna.
CHARCOT, J. M., M.D., Physician to the "Hôpital de la Salpêtrière," and Professor at the Faculty of Medicine of Paris.
CHAUVEAU, A., M.D., Professor of Physiology at the Medical School of Lyons.
DONDERS, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
VON HELMHOLTZ, H., M.D., Professor of Physiology in the University of Heidelberg.
LUDWIG, C., M.D., Professor of Physiology in the University of Leipzig.
PASTEUR, PROFESSOR L., Member of the Institute, Paris.
RINDFLEISCH, EDOUARD, M.D., Professor of Pathological Anatomy in the University of Bonn.
SIMON, SIR JOHN, K.C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington-square, W.
THIERSCH, CARL, M.D., Professor of Surgery in the University of Leipzig.
VIRCHOW, RUDOLF, M.D., Professor of Pathological Anatomy in the University of Berlin.
WILLIAMS, C. J. B., M.D., F.R.S., Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton [2, Upper Brook-street, W.].

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
T.—Treasurer.
V.-P.—Vice-President.
S.—Secretary.
C.—Member of Council.

Those marked thus (†) have paid Composition Fee for Annual Subscription.
Those marked thus (‡) have paid Composition Fee for Transactions.

GENERAL LIST OF MEMBERS.

Elected

1879 Abercrombie, John, M.D., Assistant Physician to Charing Cross Hospital, 23, Upper Wimpole-street, Cavendish-square, W.

1885 Abraham, Phineas S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 11, Nottingham-place, W.

1858 Acland, Sir Henry Wentworth, K.C.B., M.D., F.R.S., Regius Professor of Medicine, University of Oxford; Broad-street, Oxford.

1883 Acland, Theodore Dyke, M.D., Assistant Physician to St. Thomas's Hospital, and to the Hospital for Consumption, Brompton; 7, Brook-street, Hanover-square, W.

‡1866 Adams, Arthur Bayley.

1869 Adams, James Edward, Grately, Andover, Hants.

O.M. Adams, William, Consulting Surgeon to the National Orthopædic Hospital; 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

1848 Aikin, Charles A., 7, Clifton-place, Sussex-square, Hyde-park, W. (C. 1864-6.)

1872 Aikin, Charles Edmund, 7, Clifton-place, Sussex-square, Hyde-park, W.

1871 Air, A. Cummings, 2, Belle Vue, South Norwood, S.E.

1880 Aitken, Sir William, M.D., LL.D., F.R.S., Professor of Pathology, Army Medical School, Netley, Southampton; Park Villa, Weston-grove-road, Woolston, Southampton.

1869 Allbutt, Thomas Clifford, M.D., F.R.S., Consulting Physician to the Leeds General Infirmary; 6, Park-square, Leeds.

1882 Allchin, William Henry, M.B., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos-street, Cavendish-square, W.

1877 Althaus, Julius, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley-street, Cavendish-square, W.

1884 Anderson, Alexander Richard, Resident Surgeon, General Hospital, Nottingham.

1868 Anderson, J. Ford, M.D., 1, Buckland-crescent, Belsize-park, N.W.

1880 Anderson, James, M.A., M.D., C.M., 84, Wimpole-street, Cavendish-square, W.

1871 Anderson, William, Assistant Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 25, Grosvenor-road, Westminster. (C. 1888.)

1863 Andrew, James, M.D., Physician to St. Bartholomew's Hospital; 22, Harley-street, W. (C. 1868-70.)

1887 Arkle, Charles Joseph, M.D., University College Hospital.

1883 Ashby, Henry, M.D., Physician to the Manchester General Hospital for Children, and Lecturer on Diseases of Children at Owens College; 13, St. John-street, Manchester.

Elected

1882 Axe, J. Wortley, Professor of Pathological Histology and Morbid Anatomy at the Royal Veterinary College, College House, Great College-street, Camden-town, N.W.

1863 Bagshawe, Frederick, M.A., M.D., 16, Warrior-square, Hastings.

1864 Baker, William Morrant (V.-P.), Surgeon to St. Bartholomew's Hospital; 26, Wimpole-street, Cavendish-square, W. (C. 1873-6, 1881-3. S. 1878-80. V.-P. 1886-8.)

‡1856 Balding, Daniel Barley, Royston, Herts.

1880 Ball, Benjamin, Professeur à la Faculté de Médecine de Paris, Médecin en Chef de la Clinique des Maladies Mentales; rue du Faubourg St. Honoré, 3, Paris.

1881 Ballance, Charles A., M.S., Demonstrator of Anatomy, St. Thomas's Hospital; 56, Harley-street, W.

1875 Barker, Arthur E. J., Surgeon and Assistant Teacher of Clinical Surgery, University College Hospital; 87, Harley-street, Cavendish-square, W. (C. 1884-6.)

1885 Barling, Gilbert, M.B., 85, Edmund-street, Birmingham.

1874 Barlow, Thomas, M.D., B.S., Assistant Physician to University College Hospital and Physician to the Children's Hospital, Great Ormond-street; 10, Wimpole-street, Cavendish-square, W. (C. 1879-81.)

1871 Barnes, Robert, M.D., 15, Harley-street, Cavendish-square, W. (C. 1883-5.)

1862 Barratt, Joseph Gillman, M.D.

1877 Barrow, A. Boyce, Assistant Surgeon to the Westminster Hospital, and to the West London Hospital; 17, Welbeck-street, Cavendish-square, W.

1881 Barrs, Alfred George, M.D., Assistant Physician, General Infirmary, Leeds; 22, Park-place, Leeds.

1879 Bartlett, Henry, M.D., Defoe-road, Tooting-corner, S.W.

1853 Barwell, Richard, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole-street, Cavendish-square, W. (C. 1862-4.)

1861 Bastian, H. Charlton, M.A., M.D., F.R.S., Professor of Clinical Medicine in University College, and Physician to University College Hospital; 20, Queen Anne-street, W. (C. 1869-71. V.-P. 1885-87.)

1877 Bateman, Arthur W., B.A., Tenterfield, New South Wales.

†1876 Batteson, John, Medical Officer of the Royal Humane Society; 1, Coborn-place, Bow-road, E.

1882 Battle, William Henry, 6, Harley-street, W.

1870 Bäumler, Christian G. H., M.D., Professor of Materia Medica in the University of Erlangen.

1874 Beach, Fletcher, M.B., Metropolitan District Asylum, Darenth, near Dartford, Kent.

Elected

1879 Beale, Edwin Clifford, M.B., Physician to Great Northern Hospital; 23, Upper Berkeley-street, Portman-square, W.

1852 Beale, Lionel S., M.B., F.R.S., Professor of Medicine at King's College, Physician to King's College Hospital; 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

1856 Bealey, Adam, M.D., M.A., Oak-lea, Harrogate.

†1878 Beaney, James George, Senior Surgeon to the Melbourne Hospital, Melbourne, Victoria.

1870 Beck, Marcus, M.S. (V.-P.), Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole-street, Cavendish-square, W. (C. 1875-7. V.-P. 1888.)

1865 Beeby, Walter, M.D., Bromley, Kent.

†1880 Beevor, Charles Edward, M.B., 33, Harley-street, Cavendish-square. (C. 1888.)

1865 Bellamy, Edward, Senior Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 17, Wimpole street, Cavendish-square, W. (C. 1876-8.)

1883 Benham, Robert Fitzroy, Abercorn House, Baron's-court, S.W.

1847 Bennet, James Henry, M.D., Mentone, Alpes Maritimes, France.

1886 Bennett, Frederick Joseph, 28, George-street, Hanover-square, W.

1877 Bennett, William Henry, Assistant Surgeon to St. George's Hospital, Surgeon to the Belgrave Hospital for Children; 1, Chesterfield-street, Mayfair, W.

1878 Bernard, Francis R., M.D., 25, Tentercroft-street, Lincoln.

1882 Berridge, William Alfred, Redhill, Surrey.

1886 Berry, James, 60, Welbeck-street, Cavendish-square, W.

‡1856 Bickersteth, Edward R., Surgeon to the Liverpool Royal Infirmary; 2, Rodney-street, Liverpool.

1882 Bindley, Philip Henry, M.B., Fairholme, Hastings.

1878 Bindon, William John Vereker, M.D., 18, St. Ann's-street, Manchester.

1850 Birkett, Edmund Lloyd, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell-square, W.C. (C. 1856-7.)

1881 Biss, Cecil Yates, M.D., Assistant Physician to the Middlesex Hospital, Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley-street, Cavendish-square, W.

1865 Bisshopp, James, Bedford-place, Tunbridge Wells.

1877 Black, James, Assistant Surgeon to the North-West London Hospital, Lecturer on Anatomy at Westminster Hospital; 16, Wimpole-street, Cavendish-square, W.

1850 Blagden, Robert, Stroud, Gloucestershire.

1863 Blanchet, Jean B., M.D., M.S., Montreal, Quebec, Canada.

1876 Blasson, William, Edgeware, Middlesex.

1879 Boileau, J. P. H., M.D., Surgeon-Major, Army Medical Department. [India.]

Elected

1876 Bond, Thomas, M.B., Assistant Surgeon and Lecturer on Forensic Medicine to Westminster Hospital; 7, Broad Sanctuary, Westminster, S.W.

1869 Bourne, Walter, M.D., Archacho, France.

1861 Bowee, Richard Norris, Ivel Lodge, Sandy, Bedfordshire.

1881 Bowlby, Anthony A., Surgical Registrar, St. Bartholomew's Hospital; 75, Warrington crescent, Maida Vale, W. (C. 1886–8.)

1851 Bowman, Sir William, Bart., F.R.S., Consulting Surgeon to the Royal Ophthalmic Hospital; 5, Clifford-street, Bond-street, W. (C. 1855-6. V.P. 1882-4.)

1882 Boyd, Stanley, M.B., Assistant Surgeon to Charing Cross Hospital; 27, Gower-street, W.C.

1883 Bradshaw, James Dixon, M.B., 30, George-street, Hanover-square, W.

1879 Brailey, Wm. Arthur, M.D., Lecturer on Comparative Anatomy at, and Assistant Ophthalmic Surgeon to, Guy's Hospital; 11, Old Burlington-street, W.

1880 Bramwell, Byrom, M.D., 23, Drumsheugh-gardens West, Edinburgh.

1886 Brenchley, Algernon Dutton, M.B., 124, Denmark-hill, S.E.

1877 Bridges, Robert, M.B., M.A., Manor House, Yattendon, Berks.

‡1867 Bridgewater, Thomas, LL.D. Glas., M.B. Lond., Harrow-on-the-hill, Middlesex.

1873 Briggs, Jacob Myers, M.D., Coeymans, New York, U.S.

1868 Bright, G. C., M.B., Cannes, France.

1857 Briscoe, John, 12, Broad-street, Oxford.

1885 Briscoe, John F.

†1851 Bristowe, John S., M.D., F.R.S., Physician to, and Lecturer on the Theory and Practice of Medicine at, St. Thomas's Hospital; 13, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-76, 1887. P. 1885-86.)

1860 Broadbent, William Henry, M.D., Physician to St. Mary's Hospital, and Consulting Physician to the London Fever Hospital; 34, Seymour-street, Portman-square, W. (C. 1871-3. V.P. 1882-4.)

1886 Brockatt, Andrew Alexander, St. Thomas's Hospital, S.E.

Brockman, see Drake-Brockman.

1852 Brodhurst, Bernard E., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor-street, W. (C. 1862-4.)

1884 Brodie, Charles Gordon, Middlesex Hospital [Fernhill, Wootton Bridge, Isle of Wight].

1863 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield-street, Mayfair, W.

1865 Brown, Augustus, M.D., Glencoe, Hatherley-road, Sidcup, Kent.

1871 Brown, Frederick Gordon, 17, Finsbury-circus, E.C.

1875 Browne, George Buckston, 80, Wimpole-street, Cavendish-square, W.

1866 Browne, Lennox, Surgeon to the Central Throat and Ear Hospital, and to the Royal Society of Musicians; 36, Weymouth-street, Portland-place, W.

Elected

1877 Bruce, J. Mitchell, M.D., Physician to the Charing Cross Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley-street, Cavendish-square, W.

1855 Bryant, Thomas, Consulting Surgeon to Guy's Hospital; 65, Grosvenor-street, Grosvenor-square, W. (C. 1863-6. V.-P. 1877-79.)

1854 Buchanan, George, M.D., F.R.S., Medical Officer of the Local Government Board, 24, Nottingham-place, Marylebone-road, W. (C. 1864-6. V.-P. 1880-82.)

1887 Bull, William Charles, M.B., 35, Clarges-street, Piccadilly.

1878 Burnet, Robert William, M.D., 6, Upper Wimpole-street, Cavendish-square, W.

1880 Burton, Samuel Hebbert, M.B., Norfolk and Norwich Hospital, Norwich.

1887 Butler-Smythe, Albert Charles, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook-street, Grosvenor-square, W.

1872 Butlin, Henry Trentham (C.), Assistant Surgeon to, and Demonstrator of Practical Surgery and of Disease of the Larynx at, St. Bartholomew's Hospital; 82, Harley-street, Cavendish-square, W. (C. 1876-8, 1887. S. 1884-6.)

1866 Butt, William Frederick, 48, Park-street, Park-lane, W.

1883 Buxton, Dudley W., M.D., Administrator and Teacher of the use of Anæsthetics in University College Hospital; Anæsthetist to the Hospital for Women, Soho-square, and to the Dental Hospital; 82, Mortimer-street, Cavendish-square, W.

1856 Buzzard, Thomas, M.D., Physician to the National Hospital for the Epileptic and Paralysed; 56, Grosvenor-street, W. (C. 1869-70. V.-P. 1881-3.)

1885 Cahill, John, 12, Seville-street, Lowndes-square, S.W.

†O.M. Camps, William, M.D. (C. 1856-9.)

‡1855 Carpenter, Alfred, M.D., Duppas House, Croydon.

1855 Carter, H. Vandyke, M.D., Late Principal of, and Professor of Anatomy and Physiology at, Grant Medical College, Bombay.

1876 Carter, Robert Brudenell, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne-street, Cavendish-square, W.

1879 Cassidy, Joseph Lamont, M.D., 44, Harley-street, Cavendish-square, W.

1877 Casson, John Hornsey [47A, Portland-place, W.].

†1868 Cavafy, John, M.D., Physician to St. George's Hospital; 2, Upper Berkeley-street, Portman-square, W. (C. 1881-3.)

1864 Cay, Charles Vidler, Deputy Surgeon-General, Army.

1863 Cayley, William, M.D. (Treasurer), Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; 27, Wimpole-street, Cavendish-square, W. (C. 1870-1, 1875-8. S. 1872-4. V.-P. 1884-6. T. 1888.)

Elected

1869 CHAFFERS, EDWARD, Keighley, Yorkshire.

1884 CHAFFEY, WAYLAND CHARLES, M.B., Assistant Physician to the Royal Alexandra Hospital for Sick Children; 8, North-street, Brighton.

1849 CHALK, WILLIAM OLIVER, 3, Nottingham-terrace, Regent's-park, N.W (C. 1856-7.)

1876 CHARLES, T. CRANSTOUN, M.D., M.C., Lecturer on Practical Physiology at St. Thomas's Hospital; 9, Albert Mansions, Victoria-street, Westminster.

1884 CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple-row, Birmingham.

1879 CHEYNE, WILLIAM WATSON, M.B., C.M., Assistant Surgeon to King's College Hospital; 59, Welbeck-street, Cavendish-square, W. (C. 1885-7.)

1858 CHILD, GILBERT W.

1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales [care of Messrs. Dawson, 121, Cannon-street, E.C.].

1871 CHRISTIE, THOMAS BEATH, M.D., Superintendent of the Royal India Asylum, Ealing, Middlesex.

1865 CHURCH, WILLIAM SELBY, M.D., Physician to St. Bartholomew's Hospital; 130, Harley-street, Cavendish-square, W. (C. 1871-3.)

†1868 CHURCHILL, FREDERICK, M.B., Surgeon to the Victoria Hospital for Children; 4, Cranley-gardens, Queen's-gate, S.W.

1861 CLAPTON, EDWARD, M.D., 10A, St. Thomas's-street, Southwark, S.E.

1854 CLARK, Sir ANDREW, Bart., M.D., LL.D., F.R.S., Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish-square, W. (C. 1862-5. V.-P. 1881-3.)

1872 CLARK, ANDREW, Assistant Surgeon to the Middlesex Hospital; 19, Cavendish-place, W.

1886 CLARK, FRANCIS WILLIAM, Darenth Imbecile Schools, near Dartford, Kent.

1883 CLARKE, ERNEST, M.D., B.S., 21, Lee-terrace, Blackheath, S.E.

1885 CLARKE, JOHN MICHELL, M.B., 2, York Buildings, Clifton, Bristol.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon and Demonstrator of Anatomy, St. Bartholomew's Hospital; 46, Harley-street, Cavendish-square, W.

†1875 CLUTTON, HENRY HUGH, M.A., Assistant Surgeon to St. Thomas's Hospital; 2, Portland-place, W. (C. 1884-6.)

‡1865 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1885 COATS, JOSEPH, M.D., 31, Lynedoch-street, Glasgow.

1856 COCKLE, JOHN, M.D., M.A., Physician to the Royal Free Hospital; 8, Suffolk-street, Pall Mall, S.W.

1886 COLLIER, WILLIAM, M.D., 62, High-street, Oxford.

1888 COLLINS, WILLIAM JOB, M.D., M.S., 1, Albert-terrace, Regent's-park, N.W.

1879 COLLINS, WM. MAUNSELL, M.D., 10, Cadogan-place, S.W.

1878 COLLYNS, R. T. POOLE, 20, Lingfield-road, Wimbledon.

Elected

1888 Colman, Walter Stacy, M.B., West London Hospital, Hammersmith-road, W.

1882 Colquhoun, Daniel, M.D., Dunedin, New Zealand.

1882 Compton, Francis Charles, 72, High-street, Poole, Dorset.

1858 Cooke, R. T. E. Barrington, Consulting Surgeon to the Scarborough Dispensary, Consulting Surgeon to the Royal Northern Sea-Bathing Infirmary; 15, St. Nicholas-cliff, Scarborough, Yorkshire.

1871 Cooke, Thomas, Assistant Surgeon to the Westminster Hospital; 40, Brunswick-square, W.C.

1866 Coombs, Rowland Hill, Mill-street, Bedford.

1879 Cooper, Arthur, 20, Old Burlington-street, W.

1853 Cornish, William Robert, Surgeon-Major, Madras Army, Sanitary Commissioner for Madras.

1875 Cory, Robert, M.D., Assistant Obstetric Physician to St. Thomas's Hospital; 73, Lambeth Palace-road, S.E.

1876 Cottle, Wyndham, M.D., Assistant Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile-row, W.

†1861 Couper, John, Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital; 80, Grosvenor-street, Grosvenor-square, W. (C. 1870-2.)

1873 Coupland, Sidney, M.D. (Hon. Secretary), Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth-street, Portland-place, W. (C. 1878-81. S. 1886-8.)

1884 Crichton, George, M.B., 3, Cambridge-villas, Twickenham.

1873 Cripps, William Harrison, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford-place, Oxford-street, W. (C. 1883-5.)

1877 Crocker, Henry Radcliffe, M.D., Physician to the Skin Department, University College Hospital; Physician to the East London Hospital for Children; 121, Harley-street, Cavendish-square, W. (C. 1887-8.)

1856 Croft, John, Surgeon to St. Thomas's Hospital; 48, Brook-street, Grosvenor-square, W. (C. 1870-2. V.-P. 1882–4)

1879 Crooke, George Frederick, M.D., General Hospital, Birmingham.

1886 Crookshank, Edgar, M.B., Professor of Bacteriology at King's College, London; 24, Manchester-square, W.

1861 Crosby, Thomas Boor, M.D., 21, Gordon-square, W.C.

1875 Cross, Francis Richardson, 5, The Mall, Clifton, Bristol.

1885 Cullingworth, Charles James, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital; 46, Brook-street, Grosvenor-square, W.

1871 Cumberbatch, Elkin, Aural Surgeon at St. Bartholomew's Hospital; 17, Queen Anne-street, W.

1858 Cumberbatch, Laurence T., M.D., 25, Cadogan-place, Sloane-street, S.W.

Elected

1873 Cubnow, John, M.D., Professor of Anatomy at King's College, Physician to King's College Hospital, and Senior Visiting Physician to the Seamen's Hospital; 3, George-street, Hanover-square, W. (C. 1882-4.)

‡1865 Curran, William, M.D. [33, Auriol-road, West Kensington, W.], care of Mr. Lewis, Gower-street.

1884 Dakin, W. Radford, M.D., B.S., 57, Welbeck-street, Cavendish-square, W.

1884 Dallaway, Dennis, 5, Duchess-street, Portland Place, W.

1883 Dalton, Norman, M.D., Assistant Physician to King's College Hospital; 18, Clement's-inn, Strand, W.C.

1873 Davidson, Alexander, M.D., Physician to the Liverpool Royal Infirmary, Lecturer on Pathology at the Liverpool Medical School; 2, Gambier-terrace, Liverpool.

1885 Davies, Arthur, M.B., 23, Finsbury-square, E.C.

1869 Davies-Colley, J. Neville C., M.B. Surgeon to Guy's Hospital; 36, Harley-street, Cavendish-square, W. (C. 1880-82.)

1883 Davis, Edwin Harry, West Hartlepool.

‡1859 Davis, Francis William, R.N., Surgeon to the Naval Medical Establishment, Lisbon. [Agents: Messrs. Hallett and Co., 7, St. Martin's-place, Trafalgar-square, W.C.]

1879 Davy, Henry, M.D., Physician to the Devon and Exeter Hospital; 29, Southernhay, Exeter.

1866 Day, William Henry, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester-square, W.

1887 Delépine, Sheridan, M.B., 6, Chapel-place, Cavendish-square, W.

1880 Dent, Clinton T., Assistant Surgeon to St. George's Hospital; 61, Brook-street, Grosvenor-square W.

1863 Devereux, Daniel, Tewkesbury, Gloucestershire.

1856 Dick, H., M.D.

1871 Dickinson, Edward Harriman, M.A., M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Comparative Anatomy at the Liverpool School of Medicine; 162, Bedford-street, Liverpool.

1858 Dickinson, William Howship, M.D., Physician to the Hospital for Sick Children, Physician and Lecturer on Medicine to St. George's Hospital; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield-street, Mayfair, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4.)

1872 Diver, Ebenezer, M.D., Kenley, Caterham-valley, Surrey.

1872 Doran, Alban Henry Griffiths, Surgeon to Out-Patients, Samaritan Hospital; 9, Granville-place, W. (C. 1882-84.)

1888 Dove, Augustus Charles, M.B., B.S., 72, Redcliff-square, S.W.

†1866 Down, John Langdon H., M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley-street, Cavendish-square, W. (C. 1872-4.)

1872 Dowse, Thomas Stretch, M.D., 14, Welbeck-street, Cavendish-square, W.

Elected

1877 Drake-Brockman, E. F., Madras Medical Service [care of Mr. Lewis, Gower-street, W.C.]

1880 Dreschfeld, Julius, M.D., Physician to the Manchester Infirmary; 325, Oxford-road, Manchester.

1879 Drewitt, F. G. Dawtrey, M.D., 52, Brook-street, Grosvenor-square, W.

1865 Duckworth, Sir Dyce, M.D., Physician to St. Bartholomew's Hospital; 11, Grafton-street, Bond-street, W. (C. 1877.)

1863 Dudfield, Thomas Orme, M.D., 14, Ashburn-place, South Kensington, S.W.

1847 Dudgeon, Robert E., M.D., 53, Montagu-square, W.

1852 Duff, George, M.D., High-street, Elgin.

1865 Duffin, Alfred Baynard, M.D., Physician to King's College Hospital; 18, Devonshire-street, Portland-place, W. (C. 1872-4.)

1875 Duka, Theodore, M.D., Surgeon-Major, H.M.'s Bengal Army.

1868 Duke, Oliver Thomas, M.B., India.

1871 Dukes, Clement, M.D., B.S. Lond., Physician to Rugby School; Sunnyside, Rugby.

1877 Dunbar, J. J. MacWhirter, M.D., Assistant House-Physician to St. George's Hospital; Hedingham House, Clapham-common, S.W.

1877 Duncan, Andrew, M.D., 8, Henrietta-street, Covent-garden, W.C.

1880 Duncan, Jas. Matthews, M.D., LL.D., F.R.S., Obstetric Physician to St. Bartholomew's Hospital; 71, Brook-street, Grosvenor-square, W.

1884 Dunn, Louis Albert, M.B., B.S., Demonstrator of Anatomy, Guy's Hospital; and Assistant Surgeon to the East London Hospital for Children; 15, St. Thomas's-street, S.E.

1858 Durham, Arthur Edward, Surgeon to Guy's Hospital; 82, Brook-street, Grosvenor-square, W. (C. 1869-71. V.P. 1883-5.)

1879 Durham, Frederic, M.B., 82, Brook-street, Grosvenor-square, W.

†1880 Edmunds, Walter, M.D., 75, Lambeth Palace-road, S.E.

1882 Edwardes, Edward Joshua, M.D., 16, Acacia-road, St. John's Wood, N.W.

1882 Edwards, F. Swinford, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 93, Wimpole-street, Cavendish-square, W.

1883 Elder, George, M.D., Surgeon to the Hospital for Women; 17, Regent-street, Nottingham.

1867 Ellis, James, M.D., California.

1873 Engelmann, George Julius, M.D., A.M., 3003, Locust-street, St. Louis, Miss., U.S.

1846 Erichsen, John Eric, LL.D., F.R.S., Surgeon Extraordinary to Her Majesty the Queen, Emeritus Professor of Surgery at University College, and Consulting Surgeon to University College Hospital; 6, Cavendish-place, Cavendish-square, W. (C. 1849-51. V.-P. 1863-4.)

Elected

1853 Evans, Conway, M.D., The Garden House, Clement's-inn, W.C. (C. 1867-8.)

1875 Evans, Julian, A.M., M.D., Physician to the Victoria Hospital for Children; 123, Finboro'-road, Redcliffe-square, West Brompton, S.W.

1879 Eve, Frederic S., Pathological Curator of the Museum, Royal College of Surgeons of England, and Assistant Surgeon to the London Hospital; 15, Finsbury-circus, E.C. (C. 1885-7.)

1876 Ewart, James Cossar, M.B., C.M., School of Medicine, Edinburgh.

1881 Ewart, Joseph, M.D., late Professor of Medicine at Calcutta Medical College; Montpellier Terrace, Brighton.

1877 Ewart, William, M.B., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon-street, Mayfair, W.

‡1859 Ewens, John, 14, Whiteladies-road, Clifton, Bristol.

1887 Eyles, Charles Henry, Gold Coast Colony.

1872 Fayrer, Sir Joseph, K.C.S.I., M.D. F.R.S. Hon. Physician to the Queen, Surgeon-Major, Bengal Army, Examining Medical Officer to the Secretary of State for India in Council; 53, Wimpole-street, Cavendish-square, W. (C. 1880-2.)

1872 Fenn, Edward L., M.B., The Old Palace, Richmond, Surrey.

1880 Fenwick, Bedford, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest; 20, Upper Wimpole-street, Cavendish-square, W.

1883 Fenwick, E. Hurry, Assistant Surgeon to the London Hospital, Surgeon and Pathologist to St. Peter's Hospital for Stone; 5, Old Burlington-street, W.

1872 Fenwick, John C. J., M.D., Physician to the Durham County Hospital; 25, North-road, Durham.

1863 Fenwick, Samuel, M.D., Physician to the London Hospital; 29, Harley-street, W.

1885 Féré, Charles, M.D., Médecin de Bicêtre; Boulevard St. Michel, 37, Paris.

1846 Fincham, George T., M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave-road, S.W. (C. 1855.)

1876 Finlay, David W., M.D., Physician to the Middlesex Hospital; 9, Lower Berkeley-street, Portman-square, W. (C. 1886-8.)

1870 Fish, John Crockett, M.D., 92, Wimpole-street, W.

1859 Fisher, Alexander, M.D., Assistant Surgeon R.N., Her Majesty's Ship "Endymion."

1882 Fleming, George, C.B., LL.D., F.R.C.V.S., Principal Veterinary Surgeon to the Army, Cathcart Lodge, Tyrwhitt-road, St. John's, S.E.

1872 Forbes, Daniel Mackay, 204, Hoxton-street, N.

‡1866 Foster, Sir Balthazar Walter, M.D., M.P., Physician to the General Hospital, Birmingham; 16, Temple-row, Birmingham.

Elected

1872 Fotherby, Henry J., M.D., Physician to the Metropolitan Free Hospital; 3, Finsbury-square, E.C.

1880 Fowler, James Kingston, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges-street, Piccadilly, W. (C. 1887-8.)

1878 Fox, Thomas Colcott, M.B., B.A., Physician to the Skin Department of the Westminster Hospital, and to the Paddington Green Hospital; 14, Harley-street, Cavendish-square, W.

1858 Francis, Charles Richard, M.B., Bengal Medical Establishment, Indian Army.

1886 Freeman, Henry William, 24, Circus, Bath.

O.M. Frere, J. C.

1864 Frodsham, John Mill, M.D., Streatham, S.W.

1880 Gabbett, Henry Singer, M.B., 20, Burlington-place, Eastbourne.

‡1858 Gairdner, William Tennant, M.D., LL.D., Professor of Medicine in the University of Glasgow; 225, St. Vincent-street, Glasgow.

1870 Galton, John H., M.D, 39, Anerley-road, Upper Norwood, S.E.

1855 Gamgee, J.

1846 Garrod, Sir Alfred Baring, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley-street, Cavendish-square, W. (C. 1851. V.-P. 1863-5.)

1879 Garstang, Thomas Walter Harropp, The Heath, Knutsford, Cheshire.

1872 Garton, William, M.D., Hardshaw-street, St. Helen's, Lancashire.

1880 Gibbes, Heneage, M.B., Professor of Pathology, University of Michigan, Ann Arbor, Michigan, U.S.A.

1853 Gibbon, Septimus, M.D., 39, Oxford-terrace, Hyde-park, W.

1878 Gibbons, R. A., M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan-place, S.W.

1876 Gill, John, M.D., 31, Apsley-road, Clifton, Bristol.

1881 Glynn, Thomas Robinson, M.D., Physician to the Liverpool Royal Infirmary; 62, Rodney-street, Liverpool.

1873 Godlee, Rickman John, M.B., B.S. (Hon. Secretary), Assistant Surgeon to University College Hospital; Teacher of Operative Surgery in University College, London; 81, Wimpole-street, Cavendish-square, W. (C. 1877-80. S. 1887-8.)

1875 Godson, Clement, M.D., Assistant Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor-street, W.

1879 Godwin, Charles Henry Young, Surgeon Major, Army; Victoria Hospital, Netley.

1878 Golding-Bird, Cuthbert H., M.B., Assistant Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 13, St. Thomas's-street, S.E. (C. 1885-7.)

Elected

1871 Goodhart, James Frederic, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth-street, Portland-place, W. (C. 1876-8, 1886-8. S. 1883-5.)

1875 Gould, Alfred Pearce, M.S., Assistant Surgeon to the Middlesex Hospital,; 16, Queen Anne-street, W. (C. 1883-5.)

1870 Gowers, William Richard, M.D., F.R.S. (C.), Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 50, Queen Anne-street, Cavendish-square, W. (C. 1878-9.)

1858 Gowland, Peter Y., Surgeon to St. Mark's Hospital; 34, Finsbury-square, E.C.

1888 Grant, J. Dundas, M.A., M.D., C.M., 17, Finsbury Square, E.C.

1867 Green, T. Henry, M.D. (V.-P.), Physician to Charing Cross Hospital, Assistant Physician to the Hospital for Consumption, Brompton; 74, Wimpole-street, W. (C. 1871-3, 1878-9. S. 1875-6. V.-P. 1886-8.)

1873 Greenfield, William Smith, M.D., B.S., Professor of General Pathology in the University of Edinburgh; 7, Heriot-row, Edinburgh. (C. 1877-80.)

‡1855 Greenhill, William Alexander, M.D., 5, The Croft, Hastings.

1863 Greenhow, Edward Headlam, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; Castle Lodge, Reigate. (C. 1867-9. V.-P. 1877-8.)

1886 Greves, Edwin Hyla, M.D., Rodney House, Suffolk-road, Bournemouth.

1885 Griffith, Walter Spencer Anderson, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley-street, Cavendish-square, W.

1887 Griffiths, Joseph, M.B., C.M., Assistant Demonstrator of Pathology, University of Cambridge; Ponthenry, Llanellyn, S. Wales.

1876 Griffiths, Thomas D., M.D., Hearne Lodge, Swansea. [S.E.

1882 Gross, Charles M., B.S., St. Saviour's Infirmary; East Dulwich-grove,

1861 Gueneau de Mussy, Henri, M.D., 15, Rue du Cirque, Paris.

1863 Gull, Sir William Withey, Bart., M.D., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to Guy's Hospital; 74, Brook-street, Grosvenor-square, W.

1881 Gulliver, George, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck-street Cavendish-square, W.

1880 Gunn, R. Marcus, M.B., C.M., 54, Queen Anne-street, Cavendish-square, W.

1876 Gwyther, James, M.B., St. Mary Church, Torquay.

1887 Habershon, Samuel Herbert, M.D., Casualty Physician to St. Bartholomew's Hospital; 2, Upper Wimpole-street, Cavendish-square, W.

1849-59 Habershon, Samuel Osborne, M.D., 70, Brook-street, Grosvenor-square, W. (Re-elected 1874.) (C. 1855-6.)

1851 Hacon, E. Dennis, 269, Mare-street, Hackney, N.E. (C. 1872.)

1879 Hadden, Walter Baugh, M.D. (C.), Assistant Physician to, and Demonstrator of Morbid Anatomy at, St. Thomas's Hospital; 21, Welbeck-street, W. (C. 1886-8.)

Elected

1882 Haig, A., M.B., 30, Welbeck-street, Cavendish-square, W.

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

1886 Hamilton, David James, M.B., Professor of Pathology at the University of Aberdeen, 1, Albyn-place, Aberdeen.

1886 Handford, Henry, M.D., Physician to Nottingham General Hospital; 14, Regent-street, Nottingham.

1882 Harbinson, Alexander, M.D., County Lunatic Asylum, Lancaster.

1848 Hare, Charles John, M.D., Consulting Physician to University College Hospital; Berkeley House, 15, Manchester Square, W. (C. 1852-4. V.-P.1874-7.)

†1856 Harley, George, M.D., F.R.S., 25, Harley-street, Cavendish-square, W. (C. 1862-5. V.-P. 1878-80.)

1872 Harris, Henry, M.D., Trengweath-place, Redruth, Cornwall.

1879 Harris, Vincent Dormer, M.D., Demonstrator of Physiology, St. Bartholomew's Hospital, Senior Assistant Physician, Victoria Park Hospital; 31, Wimpole-street, Cavendish-square, W.

†1858 Hart, Ernest, 38, Wimpole-street, Cavendish-square, W. (C. 1867-8.)

1870 Haward, John Warrington, Surgeon to St. George's Hospital; 16, Savile-row, W. (C. 1879-81.)

1886 Hawkins, Francis Henry, M.B., Physician to St. George's and St. James's Dispensary, and to the North London Hospital for Consumption; 22, Henrietta-street, Cavendish-square, W.

1856 Heath, Christopher, Holme Professor of Clinical Surgery in University College, and Surgeon to University College Hospital; 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)

1881 Hebb, Richard G., M.D., Lecturer on Morbid Histology at the Westminster Hospital; Westminster Hospital, S.W.

1884 Hebbert, Charles Alfred, Westminster Hospital.

1878 Hellier, John B., M.B., Headingley, Leeds.

1879 Henderson, George Courtenay, M.D., Kingston, Jamaica, West Indies.

1869 Hensley, Philip J., M.D., Assistant Physician and Lecturer on Forensic Medicine, St. Bartholomew's Hospital; 4, Henrietta-street, Cavendish-square, W.

1884 Herringham, Wilmot Parker, M.B., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital, and Assistant Physician to the West London Hospital; 22, Bedford-square, W.C.

O.M. Hewett, Sir Prescott G., Bart., F.R.S., Consulting Surgeon to St. George's Hospital; Chesnut Lodge, Horsham, Sussex. (C. 1846-52. V.-P. 1854-7. *Pres.* 1863-4. V.-P. 1865-8.)

1855 Hewitt, Graily, M.D., Consulting Obstetric Physician to University College Hospital; 36, Berkeley-square, W. (C. 1865-7.)

1864 Hickman, William, M.B., Surgeon to the Samaritan Free Hospital; 1, Dorset-square, N.W.

1860 Hill, M. Berkeley, M.B., Surgeon to University College Hospital, and Surgeon for Out-Patients to the Lock Hospital; 66, Wimpole-street, Cavendish-square, W. (C. 1874-5.)

Elected

1875 Hitchcock, Harry Knight, M.D., Christowell, Branksome-park, Bournemouth, Hants.

1880 Hobson, John Morrison M.D., 65, Lower Addiscombe-road, Croydon.

1854 Holmes, Timothy, Consulting Surgeon to St. George's Hospital; 18, Great Cumberland-place, Hyde-park, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)

1850 Holt, Barnard Wight, Consulting Surgeon to the Westminster Hospital; 14, Savile-row, W. (C. 1853.)

O.M. Holthouse, Carsten, 5, Bloomsbury-sq., W.C. (C. 1852-4, V.-P. 1874-5.)

1878 Hood, Donald William Charles, M.D., Physician to the North-West London Hospital, Physician to the West London Hospital; 43, Green-street, Park Lane, W.

1864 Hood, Wharton P., M.D., 11, Seymour-street, Portman-square, W.

1870 Hope, William, M.D., 56, Curzon-street, Mayfair, W.

1882 Hopkins, John, Medical Superintendent, Central London Sick Asylum, Cleveland-street, W.

1879 Horrocks, Peter, M.D., Assistant Obstetric Physician to Guy's Hospital, 9, St. Thomas's-street, S.E.

1883 Horsley, Victor, M.B., B.S., F.R.S., Assistant Surgeon to University College Hospital, Professor of Pathology, University College, London, Superintendent of the Brown Institution, Wandsworth-road; 80, Park-street, Grosvenor-square, W. (C. 1888.)

†1880 Hovell, T. Mark, Aural Surgeon to the London Hospital; 3, Mansfield-street, Portland-place, W.

1875 Howse, Henry Greenway, M.S., Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children; 59, Brook-street, Grosvenor-square. W. (C. 1878-81.)

1884 Hudson, Charles Leopold, Pathologist and Curator of the Museum, Middlesex Hospital; Warden of the College; The College, Cleveland-st.

†1856 Hudson, John, M.D., 11, Cork-street, Bond-street, W.

1854 Hulke, John Whitaker, F.R.S., Surgeon to the Middlesex Hospital and Surgeon to the Royal London Ophthalmic Hospital; 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P. 1873-6, 1885-6. T. 1877-9. P. 1883-4.)

1853 Humby, Edwin, M.D., 83, Hamilton-terrace, St. John's Wood, N.W.

1874 Humphreys, Henry, M.D., late Physician to the Children's Hospital at Pendlebury; 16, Warrior Gardens, St. Leonards-on-Sea.

1883 Humphry, George Murray, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge.

1888 Hunter, William, M.D., St. John's College, Cambridge.

1852 Hutchinson, Jonathan, F.R.S., Consulting Surgeon to the London Hospital and to the Royal London Ophthalmic Hospital, Moorfields; 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3, 1881-3. P. 1879-80.)

1882 Hutchinson, Jonathan, junr., 15, Cavendish-square, W.

1881 Hutton, Henry Richmond, M.B., 8A, St. John-street, Manchester.

Elected

1880 Ingram, Ernest Fortescue, Newcastle, Natal, S. Africa.

1886 Jackson, Arthur Molyneux, M.B., Surrey County Asylum, Tooting, S.W.

1865 Jackson, J. Hughlings, M.D., F.R.S. (V.-P.), Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester-square, W. (C. 1872-3. V.-P. 1888.)

1886 Jackson, Philip J., 216, Great Dover-street, S.E.

1887 Jacob, Ernest Henry, M.D., 12, Park-street, Leeds.

1875 Jalland, William Hamerton, St. Leonard's House, Museum-street, York.

1888 James, J. T., 30, Harley-street, Cavendish-square, W.

‡1853 Jardine, John Lee, Capel, near Dorking, Surrey.

O.M. Jenner, Sir William, Bart., M.D., D.C.L., K.C.B., F.R.S., Consulting Physician to University College Hospital; 63, Brook-street, Grosvenor-square, W. (C. 1850-3. V.-P. 1862-4 1875-6. *Pres.* 1873-4.)

1881 Jennings, William Oscar, M.D., 8, Rue Roy, Paris.

1879 Jessop, Charles Moore, Army and Navy Club, Pall Mall, S.W.

1866 Jessop, Thomas Richard, 31, Park-square, Leeds.

1878 Johnson, Arthur Jukes, Yorkville, Ontario, Canada.

1876 Johnson, Charles Henry, late Staff Surgeon, Turkish Contingent; Winton House, Basingstoke, Hants.

O.M. Johnson, George, M.D., F.R.S., Consulting Physician to King's College Hospital; 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884-6. T. 1880-83.)

1888 Johnson, Raymond, M.B., B.S., 6, Albert-street, Regent's-park, N.W.

1881 Johnston, Joseph, M.D., Brigade Surgeon, Army Medical Department; 24, St. John's Wood-park, N.W.

1854 Johnstone, Athol A. W., St. Moritz House, 61, Dyke-road, Brighton.

1853 Jones, Sydney, M.B., Surgeon to St. Thomas's Hospital; 16, George-street, Hanover-square, W. (C. 1864-6. V.-P. 1886-7.)

1888 Jones, Talfourd, M.B., Eastbourne.

1862 Jones, Thomas Ridge, M.D., Physician to the Victoria Hospital for Children; 4, Chesham-place, S.W. (C. 1882-4.)

1858 Jones, William Price, M.D., Claremont-road, Surbiton, Kingston.

1886 Juler, Henry Edward, Assistant Surgeon Royal Westminster Ophthalmic Hospital, Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole-street, Cavendish-square, W.

1867 Kelly, Charles, M.D., Professor of Hygiene, King's College, Strand; Broadwater-road, Worthing, Sussex. (C. 1874.)

1846 Kent, Thomas J., 89, Piccadilly, W.

1852 Kershaw, W. Wayland, M.D., Kingston-on-Thames.

1879 Kesteven, William Henry, 16, Parkhurst-road, Holloway, N.

1859 Kiallmark, Henry Walter, 5, Pembridge-gardens, Bayswater, W. (C. 1875-6.)

1882 Kidd, Percy, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook-street, Grosvenor-square, W.

Elected

1867 King, Edwin Holborow, Netley Court, Southampton.

1871 King, Robert, M.B., Bargaly, Newton Stewart, N.B.

1852 Kingdon, J. Abernethy, Surgeon to the City Dispensary, and to the City of London Truss Society; 2, New Bank-buildings, Lothbury, E.C.

1878 Klein, Edward Emanuel, M.D., F.R.S., Joint Lecturer on Physiology at St. Bartholomew's Hospital; 19, Earl's Court-sq., S.W. (C. 1888.)

1875 Lacy, C. S. De Lacy, M.B.

1878 Lancereaux, Etienne, M.D., 44, Rue de la Bienfaisance, Paris.

1882 Lane, William Arbuthnot, M.B., M.S., Assistant Surgeon to Guy's Hospital, and to the Hospital for Sick Children, Great Ormond-street; 14, St. Thomas's-street, S.E.

1865 Langton, John, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital, and Surgeon to the City of London Truss Society; 2, Harley-street, Cavendish-square, W. (C. 1882-4.)

1886 Lankester, Herbert Henry, M.D., 1, Elm-park-gardens, South Kensington, S.W.

1869 Larcher, O., M.D. Par., Laureate of the Institute of France, of the Medical Faculty and Academy of Paris; 97, Rue de Passy, Paris. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longmans.]

1884 Larder, Herbert, Medical Superintendent, Whitechapel Infirmary, E.

1873 Latham, Peter Wallwork, M.D., Physician to Addenbrooke's Hospital, and Downing Professor of Medicine, Cambridge University; 17, Trumpington-street, Cambridge.

1876 Law, William Thomas, M.D., 9, Norfolk-crescent, Hyde-park, W.

1883 Lawford, John Bowring, M.D., C.M., Assistant Ophthalmic Surgeon to St. Thomas's Hospital; 6, Upper Wimpole-street, W.

1853 Lawrence, Henry John Hughes, Picton House, Llandowror, St. Clears. (C. 1873-5.)

1887 Lawson, Bernard, 12, Harley-street, Cavendish-square, W.

1859 Lawson, George, Surgeon Oculist to H.M. the Queen, Surgeon to the Middlesex Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 12, Harley-street, Cavendish-square, W. (C. 1870-1. V.-P. 1884-5.)

1879 Laycock, George Lockwood, M.B., 42, Manchester-street, Manchester-square, W.

1875 Lediard, Henry Ambrose, M.D., Surgeon to the Cumberland Infirmary 41, Lowther-street, Carlisle.

1852 Lee, Henry, Consulting Surgeon to St. George's Hospital; 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)

1879 Leech, Daniel John, M.D., 96, Mosley-street, Manchester.

1877 Lees, David B., M.D., Physician to St. Mary's Hospital, and Assistant Physician to the Hospital for Sick Children; 22, Weymouth-street, Portland-place, W.

Elected

1867 Lees, Joseph, M.D., 21, Brixton-road, S.W.

1877 Leeson, John Rudd, M.D., C.M., 6, Clifden-road, Twickenham.

1868 Legg, John Wickham, M.D., Hotel Bourséjour, Cannes. (C. 1874-5.)

1884 Leonard, Henry James, M.B., 279, Camden-road, N.

1861 Lichtenberg, George, M.D., 47, Finsbury-square, E.C.

1875 Lingard, Alfred, 49, Palace-road, Albert Embankment, S.E.

1877 Lister, Sir Joseph, Bart., D.C.L., LL.D., F.R.S. (V.-P.), Professor of Clinical Surgery at King's College, and Surgeon to King's College Hospital; 12, Park-crescent, Regent's-park, W. (C. 1880-2. V.-P 1887-8.)

1878 Littlejohn, Salter G., M.B., C.M., Central London District Schools, Hanwell.

†1862 Little, Louis S., China. [18, Park-street.]

1874 Liveing, Edward, M.D., 52, Queen Anne-street, Cavendish-square, W.

1863 Liveing, Robert, M.D., Consulting Physician to the Skin Department at the Middlesex Hospital; 11, Manchester-square, W. (C. 1876.)

1882 Lockwood, C. B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 19, Upper Berkeley-street, W.

1876 Longhurst, Arthur Edwin Temple, M.D., 22, Wilton-street, Grosvenor-place, S.W. (C. 1885-7.)

1881 Lubbock, Montagu, M.D., Assistant Physician to Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond-street; 19, Grosvenor-street, W.

1873 Lucas R. Clement, M.B., M.S., Senior Assistant Surgeon to Guy's Hospital, and Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury-square, E.C. (C. 1883-5.)

1880 Lund, Edward, Consulting Surgeon to the Royal Infirmary; 22, St. John-street, Manchester.

1879 Lunn, John Reuben, Resident Medical Officer, New Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, Notting-hill, W.

1887 Lyon, Thomas Glover, M.D., 39, King-street, Cheapside, E.C.

1871 MacCarthy, Jeremiah, M.A., Surgeon to the London Hospital; 15, Finsbury-square, E.C. (C. 1878-80.)

1873 McConnell, J. F., Professor of Pathology, Medical College, Calcutta. [Per Grindlay & Co., Parliament-street.] 14, St. James's-square, S.W.

1871 Mac Cormac, Sir William, Surgeon to St. Thomas's Hospital; 13, Harley-street, W. (C. 1878-80.)

1873 Mackellar, Peter H., M.B., Medical Officer, Fever Hospital, Landor-road, Stockwell, S.W.

1882 Mackenzie, Frederic Morell, 29, Hans-place, S.W.

1870 Mackenzie, George Welland, 13, William-street, Lowndes-square, S.W.

1885 Mackenzie, Hector Gavin, St. Thomas's Hospital, S.E.

1870 Mackenzie, John T., Bombay, India. [East India United Service Club, 14, St. James's-square.]

Elected

1864 Mackenzie, Sir Morell, M.D., Physician to the Hospital for Diseases of the Throat, and Lecturer on Diseases of the Throat at the London Hospital; 19, Harley-street, Cavendish-square, W.

1878 Mackenzie, Stephen, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 26, Finsbury-square, E.C. (C. 1888.)

1879 Maclagan, Thomas John, M.D., 9, Cadogan-place, Belgrave-square, S.W.

1865 MacLaurin, Henry Normand, M.D., 187, Macquarie-street, Sydney, New South Wales.

1879 MacMahon, James Thomas, 25, West-hill, Dartford, Kent.

1876 Macnamara, Charles, Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor-street, W.

1879 Macready, Jonathan Forster, 51, Queen Anne-street.

1885 Maguire, Robert, M.D., 4, Seymour-street, Portman-square, W.

1877 Makins, George Henry, Assistant Surgeon to the Evelina Hospital for Children; 2, Queen-street, May Fair, W.

1887 Malcolm, John David, M.B., C.M., Surgeon in charge of out-patients, Samaritan Free Hospital; 24, Bryanston-street, Portman-square, W.

1876 Mallam, Benjamin, Rose Bank, Blackall-road, Exeter.

1876 Maples, Reginald, Kingsclere, near Newbury.

1868 Marsh, F. Howard, Assistant Surgeon to St. Bartholomew's Hospital, Surgeon to the Hospital for Sick Children; 36, Bruton-street, Berkeley-square, W. (C. 1876-7.)

1846 Marshall, John, F.R.S., Consulting Surgeon to University College Hospital, 10, Savile-row, W. (C. 1861.)

1856 Martin, Robert, M.D., Consulting Physician to St. Bartholomew's Hospital; 51, Queen Anne-street, Cavendish-square, W. (C. 1871-2.)

1887 Martin, Sidney, M.D., B.S., 60, Gower-street, W.C.

1867 Mason, Philip Brookes, Burton-on-Trent.

1884 Maudsley, Henry Carr, M.D., 11, Spring-street, Melbourne, Victoria.

†1852 May, George, Jun., M.B., Surgeon, Royal Berkshire Hospital, Reading.

1888 May, W. P., M.B., B.S., Goldburn, Eltham Road, Blackheath.

1881 Maylard, Alfred Ernest, M.B., Lecturer on Anatomy, Western Medical School, Glasgow; 4, Berkeley-terrace, Glasgow.

1874 Meredith, William Appleton, M.B., Surgeon to the Samaritan Hospital; 6, Queen Anne-street, Cavendish-square, W.

1859 Messer, John Cockburn, M.D., Assistant Surgeon R.N.

1866 Mickley, George, M.A., M.B., St. Luke's Hospital, Old-street, E.C.

1877 Milner, Edward, Surgeon to the Lock Hospital; 32, New Cavendish-street, Portland-place, W.

1882 Money, Angel, M.D., Assistant Physician to University College Hospital, and to the City of London Hospital for Disease of the Chest, Victoria-park; 24, Harley-street, Cavendish-square, W.

1879 Moore, Norman, M.D., Assistant Physician to St. Bartholomew's Hospital, and Demonstrator of Morbid Anatomy and Warden of the College; The College, St. Bartholomew's Hospital. (C. 1885-7.)

1881 Moore, Thomas, 6, Lee-terrace, Blackheath, S.E.

Elected

1847 Morgan, John, 3, Sussex-place, Hyde-park-gardens, W. (C. 1856-8.)

1875 Morgan, John H., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond-street; 68, Grosvenor-street, W. (C. 1886-8.)

1874 Morison, Alexander, M.D., C.M., Dunnottar, 115, Green-lanes, Highbury, N.

1880 Morison, Basil Gordon, M.B., C.M., 70, Marquess-road, Canonbury, N.

1869 Morris, Henry, M.A., M.B. (V.-P.), Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield-street, Portland-place, W. (C. 1877-9, 1884-6. S. 1881-3. V.-P. 1888.)

1879 Morris, Malcolm Alexander, Surgeon to the Skin Department at St. Mary's Hospital; 8, Harley-street, Cavendish-square, W.

1875 Morton, John, M.B., Guildford.

1884 Mott, Frederick Walker, M.D., Lecturer on Physiology, Charing Cross Hospital; Meadowlead, Gayton-road, Harrow.

1879 Moullin, Charles W. Mansell, M.D., Assistant Surgeon to the London Hospital; 69, Wimpole-street, Cavendish-square, W.

1878 Mumford, William Lugar, M.D., 1, Bartlett's-passage, Holborn-circus, E.C.

1876 Munro, William, M.D., C.M., 102, Carl-street, Lower Broughton-road, Manchester.

1885 Murray, Hubert Montague, M.D., Assistant Physician to, and Lecturer on Pathology and Morbid Anatomy at, Charing Cross Hospital, 27, Savile-row, W.

1864 Myers, Arthur B. R., Surgeon to 1st Battalion Coldstream Guards; (C. 1872-3.)

1882 Myers, A. T., M.D., Medical Registrar, St. George's Hospital; 9, Lower Berkeley-street, Portman-square, W.

1887 Myers, W. H., Fort Wayne, Indiana, U.S.A.

1874 Nankivell, Arthur Wolcot, St. Bartholomew's Hospital, Chatham.

1887 Nason, Edward Noel, M.B., London Hospital, Whitechapel-road, E.

1873 Nettleship, Edward, Ophthalmic Surgeon to St. Thomas's Hospital, and Assistant Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole-street, Cavendish-square, W. (C. 1882-4.)

1875 Newby, Charles Henry, 15, Landport-terrace, Southsea, Hants.

1865 Newman, William, M.D., Stamford, Lincolnshire.

1868 Nicholls, James, M.D., Chelmsford, Essex.

1876 Nicholson, John Francis, M.D., Physician to the Hull General Infirmary; 29, Albion-street, Hull.

1878 Nootт, W. M., 8, Kensington-park-road, W.

1864 Norton, Arthur T., Surgeon to St. Mary's Hospital; 101, Harley-street, Cavendish-square, W. (C. 1877-9.)

1883 Norvill, Frederic Harvey, M.B., Summerland, Yeovil, Somersetshire.

1856 Nunn, Thomas William, Consulting Surgeon to the Middlesex Hospital; 8, Stratford-place, Oxford-street, W. (C. 1864-6. V.-P. 1878-80.)

Elected

1871 Nunneley, Rev. Frederick Barham, M.D.

1880 O'Connor, Bernard, M.D., Physician to the North London Consumption Hospital, and Physician to the Westminster General Dispensary; Greenhill-park, Harlesden, N.W.

1873 O'Farrell, George Plunkett, M.B., Montenotte House, Cork, Ireland

1880 Ogilvie, George, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 13, Welbeck-street, Cavendish-square, W.

1880 Ogilvie, Leslie, M.B., Physician to the Children's Hospital, Paddington-green; 46, Welbeck-street, Cavendish-square, W.

1850 Ogle, John W., M.D., Consulting Physician to St. George's Hospital; 30, Cavendish-sq., W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)

1876 Oliver, John Ferens, M.D., 12, Old Elvet, Durham.

1888 Openshaw, Thomas Horrocks, M.B., B.S., 21, Gower-street, W.C.

1860 Orange, William, M.D., Broadmoor, Wokingham, Berkshire.

1875 Ord, William Miller, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook-street, Grosvenor-square W. (C. 1880-2.)

1879 Ormerod, J. A., M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen-square, and to the City of London Hospital for Diseases of the Chest, Victoria-park; 25, Upper Wimpole-street, W. (C. 1887-8.)

1881 Owen, Isambard, M.D., Assistant Physician to St. George's Hospital; 5, Hertford-street, Mayfair, W.

1865 Owles, James Allden, M.D., 106, Philbeach-gardens, South Kensington.

1875 Page, Herbert William, M.A., M.C. Cantab., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley-street Cavendish-square, W.

1870 Paget, Sir James, Bart., D.C.L., LL.D., F.R.S. (President), Consulting Surgeon to St. Bartholomew's Hospital; 1, Harewood-place, Hanover-square, W. (P. 1887-8.)

1884 Paget, Stephen, 57, Wimpole-street, Cavendish-square, W.

1872 Parker, Robert William, Surgeon to the East London Children's Hospital; 8, Old Cavendish-street, W. (C. 1881-3.)

1874 Parker, Rushton, M.B., B.S., Professor of Surgery in University College, Liverpool, and Assistant Surgeon to the Royal Infirmary; 59, Rodney-street, Liverpool.

1853 Parkinson, George, 50, Brook-street, Grosvenor-square, W.

1882 Pasteur, William, M.D., Medical Registrar to the Middlesex Hospital; 19, Queen-street, Mayfair, W.

1885 Paul, Frank Thomas, 44, Rodney-street, Liverpool.

1865 Pavy, Frederick William, M.D., F.R.S., Senior Physician to Guy's Hospital; 35, Grosvenor-street, W. (C. 1872-4.)

Elected

1868 Payne, Joseph Frank, M.D. (V.-P.), Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole-street, Cavendish-square, W. (C. 1873-5, 1883-5. S. 1880-2. V.-P. 1888.)

1872 Pearce, Joseph Chaning, M.D., C.M., Montague House, St. Lawrence-on-Sea, Kent.

1878 Pearse, Thomas Frederick, M.D., 10, Montague-street, Russell-square, W.C.

1863 Pearson, David R., M.D., 23, Upper Phillimore-place, Kensington, W.

1884 Pedley, F. Newland, 32, Devonshire-place, Portland-place, W.

1879 Peel, Robert, 130, Collins-street East, Melbourne, Victoria.

1887 Penrose, Francis George, M.D., Colebyfield, Wimbledon.

1884 Pepper, Augustus Joseph, M.B., C.M., Surgeon to St. Mary's Hospital; 13, Wimpole-street, Cavendish-square, W.

1888 Perry, Edwin Cooper, M.B., 24, Railway Approach, London Bridge, S.E.

1878 Philipps, Sutherland Rees, M.D., St. Ann's-heath, Virginia-water, Chertsey.

1871 Phillips, Charles Douglas F., M.D., 10, Henrietta-street, Cavendish-square, W.

1878 Phillips, John Walter, Physician to the Benevolent Asylum of Melbourne; 30, Stanley-street, West Melbourne, Victoria.

1875 Philpot, Harvey John, 14, Finsbury-circus, E.C., and 74, Sutherland-avenue, Maida-vale, W.

1863 Pick, Thomas Pickering, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 18, Portman-street, Portman-square, W. (C. 1870-1. V.-P. 1885-7.)

1867 Pitt, Edward G., M.D.

1884 Pitt, George Newton, M.D., Assistant Physician and Pathologist at Guy's Hospital; 10, St. Thomas's-street, Southwark, S.E.

1876 Pitts, Bernard, M.A., M.B., Assistant Surgeon to St. Thomas's Hospital; 31, Harley-street, Cavendish-square, W. (C. 1888.)

1887 Pitts, Robert Zaccheus, Springfield, Chelmsford.

1886 Pocock, Walter, 374, Brixton-road, S.W.

1883 Poland, John, Demonstrator of Anatomy at Guy's Hospital; 16, St. Thomas's-street, Southwark, S.E.

1882 Pollard, Bilton, Assistant Surgeon and Surgical Registrar, University College Hospital; 24, Harley-street, Cavendish-square, W.

1846 Pollock, George D. (Trustee), Consulting Surgeon to St. George's Hospital; 36, Grosvenor-street, W. (S. 1850-3. C. 1854-6. V.-P. 1863-5. P. 1875-6.)

1850 Pollock, James Edward, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 52, Upper Brook-street, W. (C. 1862-4. V.-P. 1879-81.)

1870 Poore, George Vivian, M.D., Physician to University College Hospital; 30, Wimpole-street, W. (C. 1883-5.)

Elected

1876 Port, Heinrich, M.D., 48, Finsbury-square, E.C.

1879 Potter, Henry Percy, St. Mary Abbotts' Infirmary, Marloes-road, Kensington, W.

1881 Powell, Henry Albert, M.A., Elm Cottage, Beckenham.

1866 Powell, Richard Douglas, M.D. (V.-P.), Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital, Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole-street, Cavendish-square, W. (C. 1873-5, 1881-3. S. 1877-9. V.-P. 1887-8.)

1865 Power, Henry, Ophthalmic Surgeon to St. Bartholomew's Hospital; 37A, Great Cumberland-place, Hyde-park, W. (C. 1876-7.)

1884 Power, D'Arcy, M.B., Curator of St. Bartholomew's Hospital; 26, Bloomsbury-square, W.C.

1887 Pratt, William Sutton, Weedon, Northamptonshire.

1884 Price, J. A. P., M.D., 41, Castle-street, Reading.

1888 Priestley, R. C., M.B., 65, Upper Berkeley-street, W.

1856 Priestley, William Overend, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford-street, Mayfair, W.

1888 Primrose, Alexander, M.B., C.M.

1882 Pringle, J. J., M.B., Assistant Physician to, and Physician in Charge of Skin Department at, the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton-street, Mayfair, W.

†1848 Purnell, John James, Surgeon to the Royal General Dispensary; Woodlands, Streatham-hill, S.W. (C. 1858-61.)

1865 Pye-Smith, Philip Henry, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 54, Harley-street, Cavendish-square, W. (C. 1874-7.)

O.M. Quain, Richard, M.D., F.R.S. (Trustee), Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 67, Harley-street, Cavendish-square, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1884 Rake, Beaven Neave, M.D., Government Medical Officer, and Medical Superintendent of the Leper Asylum, Trinidad.

1872 Ralfe, Charles Henry, M.D., M.A., Assistant Physician to the London Hospital; 26, Queen Anne-street, W. (C. 1877-9.)

1857 Ramskill, J. Spence, M.D., Consulting Physician to the London Hospital, Physician to the National Hospital for the Paralysed and Epileptic; 5, St. Helen's-place, Bishopsgate-street, E.C.

1848 Randall, John, M.D., Medical Officer, St. Marylebone Infirmary; 204, Adelaide-road, N.W. (C. 1864-6.)

1857 Ranke, Henry, M.D., Munich.

Elected

1865 Rasch, Adolphus A., M.D., Physician for Diseases of Women to the German Hospital; 7, South-street, Finsbury-square, E.C.

1887 Raven, Thomas Francis, Broadstairs, Kent.

1870 Ray, Edward Reynolds, Dulwich, S.E.

1871 Rayner, Henry, M.D., Lecturer on Mental Diseases at St. Thomas' Hospital, Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

1858 Reed, Frederick George, M.D., 46, Hertford-street, Mayfair, W.

1866 Reeves, Henry Albert, Assistant Surgeon to the London Hospital; 7, Grosvenor-street, W.

1875 Reid, Robert William, M.D., C.M., Lecturer on Anatomy at St. Thomas's Hospital; 77, Lambeth Palace-road, S.E.

1881 Renner, William, M.R.C.S., Wilberforce-street, Free Town, Sierra Leone.

1854 Reynolds, J. Russell, M.D., F.R.S., Consulting Physician to University College Hospital; 38, Grosvenor-street, W. (C. 1868-9.

1871 Richards, J. Peeke, Medical Superintendent, Middlesex County Lunatic Asylum, Hanwell, W.

1888 Ricketts, Edwin, M.D., Portsmouth.

1866 Rivington, Walter, M.S. Lond., Surgeon to the London Hospital; 22, Finsbury-square, E.C.

‡1865 Roberts, David Lloyd, M.D., Physician to St. Mary's Hospital, Manchester; 11, St. John's-street, Manchester.

1871 Roberts, Frederick Thomas, M.D., Professor of Materia Medica at University College, and Physician to University College Hospital and to the Hospital for Consumption, &c., Brompton; 102, Harley-street, Cavendish-square, W. (C. 1883-5.)

1878 Roberts, William Howland, M.D., Surgeon, Madras Army, Madras [East India United Service Club, St. James's Square].

1885 Robinson, Arthur Henry, M.D., Mile End Infirmary, Bancroft-road, E.

1887 Robinson, Henry Betham, M.B., 7, York-crescent, Lower Norwood, S.E.

1882 Robinson, Tom, M.D., 9, Princes-street, Cavendish-Square, W.

1882 Roeckel, Waldemar Joseph, 12, Upper Berkeley-street, Portman-square, W.

1888 Rolleston, H. D., M.B., Cambridge.

1858 Rose, Henry Cooper, M.D., Surgeon to the Hampstead Dispensary, Penrose House, Hampstead, N.W. (C. 1873-4.)

1876 Rose, William, M.B., B.S., Professor of Surgery at King's College, Surgeon to King's College Hospital; 50, Harley-st., Cavendish-sq., W.

1879 Ross, James, M.D., C.M., 335, Oxford-street, Manchester.

1875 Rossiter, George Frederick, Cairo Lodge, Weston-super-Mare.

1877 Roth, Bernard, 29, Queen Anne-street, Cavendish-square, W., and Rossmore, Preston-road, Brighton.

1858 Rouse, James, Surgeon to St. George's Hospital; 2, Wilton-street, Grosvenor-place, S.W.

Elected

1881 Routh, Amand Jules McConnel, M.D., B.S., Assistant Physician Accoucheur to the Charing Cross Hospital, and Physician to the Samaritan Free Hospital; 6, Upper Montagu-street, W.

*1887 Roy, Charles Stuart, M.D., Professor of Pathology, Cambridge.

1869 Rutherford, William, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas-crescent, Edinburgh.

1882 Sainsbury, Harrington, M.D., Assistant Physician and Pathologist to the Royal Free Hospital, 63, Welbeck-street, W.

1853 Salter, S. James A., M.B., F.R.S. Late Dental Surgeon to Guy's Hospital; Basingfield, near Basingstoke, Hants. (C. 1861-3. V.-P. 1880-2.)

1852 Sanderson, Hugh James, M.D., 26, Upper Berkeley-street, Portman-square, W.

1854 Sanderson, John Burdon, M.D., F.R.S., Waynflete Professor of Physiology at the University of Oxford; 50, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)

1875 Sangster, Charles, 148, Lambeth-road, S.E.

1877 Sankey, H. R. O., County Asylum, Prestwich, Manchester.

‡1847 Sankey, W. H. Octavius, M.D., Boreatton-park, Shrewsbury. (C. 1855.)

1886 Saundby, Robert, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; 83A, Edmund-street, Birmingham.

1871 Saunders, Charles Edward, M.D., Sussex County Lunatic Asylum, Hayward's Heath.

1873 Savage, George Henry, M.D., Bethlem Royal Hospital, St. George's-road, S.E. (C. 1881-3.)

1882 Savill, Thomas Dixon, M.D., Paddington Infirmary, Harrow-road, W.

1877 Semon, Felix, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole-street, Cavendish-square, W. (C. 1885-7.)

1852 Semple, Robert Hunter, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington-square, W.C. (C. 1859-61.)

1872 Sergeant, Edward, Medical Officer of Health, Town Hall, Bolton, Lancashire.

1876 Sharkey, Seymour J., M.D., Assistant Physician and Demonstrator of Morbid Anatomy to St. Thomas's Hospital; 2, Portland-place, W. (C. 1884-6.)

1880 Shattock, S. G., Demonstrator of Surgical Pathology, and Curator of Museum, St. Thomas's Hospital; 4, Crescent-road, the Downs, Wimbledon. (C. 1885-7.)

1885 Shaw Lauriston Elgie, M.D., Medical Registrar and Demonstrator of Practical Medicine, Guy's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria-park; 15, St. Thomas's-street, Southwark, S.E.

1884 Sheild, Arthur Marmaduke, M.B., B.S., Assistant Surgeon to the Charing Cross Hospital; 20, Stratford-place, Oxford-street, W.

1886 Sherrington, C. S., M.B., Gonville and Caius College, Cambridge.

Elected

1856 Shillitoe, Buxton, Surgeon to the Great Northern Hospital, and to the Lock Hospital; 2, Frederick's-place, Old Jewry, E.C.

1855 Sibley, Septimus W., 7, Harley-street, Cavendish-square, W. (C. 1863-5. V.-P. 1879-81.)

1887 Sibley, Walter Knowsley, 7, Harley-street, Cavendish-square, W.

1875 Siddall, Joseph Bower, M.D., C.M. [care of G. Siddall, Esq., Matlock, Derbyshire].

1880 Silcock, A. Quarry, M.D., B.S., Surgeon in charge of Out-patients, St. Mary's Hospital, and Assistant Surgeon, Royal London Ophthalmic Hospital, Moorfields; 101, Harley-street, Cavendish-square, W. (C. 1888.)

O.M. Simon, Sir John, K.C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington-square, W. (C. 1846-8. V.-P. 1855-9. *Pres.* 1867-8. V.-P. 1869-71.)

1866 Sims, Francis Manley Boldero, 12, Hertford-street, Mayfair, W.

1877 Skinner, William A., 45, Upper Belgrave-street, S.W.

1887 Smallpeice, William Donald, 42, Queen Anne's-gate, S.W.

1875 Smee, Alfred Hutchinson, The Grange, Hackbridge, Carshalton, Surrey.

1879 Smith, E. Noble, Senior Surgeon, and Surgeon to the Orthopædic Department of the Farringdon Dispensary, and Orthopœdic Surgeon to the British Home for Incurables; 24, Queen Anne-street, Cavendish-square.

1887 Smith, Frederick John, M.B., 7, West-street, Finsbury-circus, E.C.

1875 Smith, George John Malcolm, M.B., Hurstpierpoint, Sussex.

1872 Smith, Gilbart, M.D., Assistant Physician to the London Hospital, Physician to the Royal Hospital for Diseases of the Chest, City-road, Visiting Physician to the Margaret-street Infirmary for Consumption; 68, Harley-street, Cavendish-square, W.

1863 Smith, Henry, Emeritus Professor of Surgery at King's College, London; 83, Wimpole-street, Cavendish-square, W. (C. 1873-4.)

1878 Smith, Herbert Urmson, M.B., Cape Colony.

1846 Smith, Protheroe, M.D., Physician to the Hospital for Women; 42, Park-street, Grosvenor-square, W.

1873 Smith, Richard T., M.D., Physician to the St. Pancras Dispensary; 53, Haverstock-hill, N.W.

1883 Smith, Robert Percy, M.D., Bethlem Royal Hospital, St. George's Road, S.E.

1869 Smith, Robert Shingleton, M.D., Lecturer on Physiology, Bristol Medical School; 9, Richmond-hill, Clifton, Bristol.

1856 Smith, Thomas, Surgeon to St. Bartholomew's Hospital; 5, Stratford-place, Oxford-street, W. (C. 1867-9. V.-P. 1877-8.)

1866 Smith, William, Melbourne, Australia.

1870 Smith, William Johnson, Surgeon, Seamen's Hospital, Greenwich, S.E. (C. 1879-81.)

1869 Smith, William Wilberforce, M.D., 14, Stratford-place, W.

Elected

1870 Snow, William Vicary, M.D., Richmond Gardens, Bournemouth.

1868 Southey, Reginald, M.D., Commissioner in Lunacy; 32, Grosvenor-road, Pimlico. (C. 1882-4.)

1887 Spencer, Walter George, M.B., Assistant Surgeon to Westminster Hospital; 94, Wimpole-street, Cavendish-square, W.

1888 Spicer, Robert Henry Scanes, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck-street, Cavendish-square, W.

1868 Spry, G. Frederick Hume, M.D., Surgeon-Major 2nd Life Guards, Regent's-park Barracks, N.W.

1861 Squire, Alexander Balmanno, M.B., 24, Weymouth-street, Portland-place, W.

1885 Squire, John Edward, M.D., Physician to North London Consumptive Hospital, 23, Seymour-street, Portman-square, W.

1854 Stewart, William Edward, 16, Harley-street, Cavendish-square, W.

1879 Stirling, Edward Charles, Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex-street, Strand, W.C.].

1883 Stoker, George, Surgeon for Out-patients, Hospital for Diseases of the Throat and Chest, Golden-square; 14, Hertford-street, Mayfair, W.

1881 Stokes, Henry Fraser, 2, Highbury-crescent, N.

1884 Stonham, Charles, Curator of the Anatomical Museum, University College, London, and Assistant Surgeon to the Westminster Hospital; 62, Welbeck-street, Cavendish-square, W.

1875 Sturge, W. A., M.D., Maison Malausséna, 27, Boulevard Dubouchage, Nice

†1871 Sutherland, Henry, M.D., 6, Richmond-terrace, Whitehall, S.W.

1864 Sutton, Henry G., M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury-square, E.C. (C. 1875-6.)

1882 Sutton, John Bland, Assistant Surgeon to, and Lecturer on Comparative Anatomy at, the Middlesex Hospital; 22, Gordon-street, Gordon-square, W.C. (C. 1887-8.)

‡1867 Swain, William Paul, 17, The Crescent, Plymouth.

1881 Symonds, Charters James, M.S. (C.), Assistant Surgeon to Guy's Hospital, and to the Evelina Hospital for Sick Children; 26, Weymouth-street, Portland-place, W. (C. 1886-8.)

1870 Tait, Robert Lawson, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham.

1886 Targett, James Henry, M.B., M.S., Assistant Pathologist, College of Surgeons, Guy's Hospital, S.E.

1864 Tatham, John, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 12, George-st., Hanover-sq., W.

1870 Tay, Waren, Surgeon to, and Demonstrator of Practical Anatomy at, the London Hospital; 4, Finsbury-square, E.C. (C. 1881-2.)

1885 Taylor, Henry H., Hospital for Consumption, Brompton, S.W.

1871 Taylor, Frederick, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, and Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's-street, S.E. (C. 1879-81.)

Elected

1880 Taylor, Seymour, M.D., M.C., Physician to the North London Hospit for Consumption; 16, Seymour-street, Portman-square, W.

1879 Thin, George, M.D., 22, Queen Anne-street, Cavendish-square, W.

1870 Thomas, John Davies, M.B., Adelaide, South Australia [care of Mr. H. K. Lewis, Gower-street].

1852 Thompson, Sir Henry, Knt., Emeritus Professor of Clinical Surgery in University College; 35, Wimpole-street, Cavendish-square, W. (S. 1859-63. C. 1865-67. V.-P. 1868-70.)

1884 Thomson, John, M.B., C.M., 18, Walker-street, Edinburgh.

1874 Thornton, John Knowsley, M.B., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman-street, Portman-square, W.

1872 Thornton, William Pugin, 35, St. George's-road, Canterbury.

1865 Thorowgood, J. C., M.D., Lecturer on Materia Medica at the Middlesex Hospital, Physician to the City of London Hospital for Diseases of the Chest; 61, Welbeck-street, W. (C. 1876-78.)

1880 Tirard, Nestor Isidore, M.D., Professor of Materia Medica at King's College, London; Assistant Physician, King's College Hospital, and to the Evelina Hospital for Sick Children; 28, Weymouth-street, Portland-place.

1884 Tivy, William James, 8, Lansdowne-place, Clifton, Bristol.

1856 Tomes, Sir John, F.R.S., Consulting Dental Surgeon to the Middlesex Hospital; Upwood Gorse, Caterham, Surrey. (C. 1867-9.)

1864 Tonge, Morris, M.D., Harrow-on-the-hill, Middlesex.

1882 Tooth, H. H., M.D., Assistant Demonstrator of Practical Physiology, St. Bartholomew's Hospital; 34, Harley-street, Cavendish-square, W.

1886 Totsuka, Kankai, 65, Lambeth Palace-road, S.E.

1872 Townsend, Thomas Sutton, 68, Queen's Gate, South Kensington, S.W.

1888 Trevelyan, E. F. M.D., Seamen's Hospital, Greenwich, S.E.

1881 Treves, Frederick, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole-street, Cavendish-square, W. (C. 1887-8.)

1851 Trotter, John W., Surgeon-Major, Coldstream Guards; Bossall Vicarage, York. (C. 1865-9.)

1859 Truman, Edwin Thomas, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington-street, W.

1888 Tubby, Alfred Herbert, M.B., 63, Studley-road, Clapham.

1867 Tuckwell, Henry Matthews, M.D., late Physician to the Radcliffe Infirmary; 64, High-street, Oxford.

1858 Tudor, John, Dorchester, Dorset.

1875 Turner, Francis Charlewood, M.D., Physician to the London Hospital; 15, Finsbury-square, E.C. (C. 1884-6.)

1882 Turner, George Robertson, Visiting Surgeon, Seamen's Hospital, Greenwich; Assistant Surgeon to, and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green-street, Park-lane.

Elected.

1863 Turner, James Smith, Consulting Dental Surgeon to the Middlesex Hospital; 12, George-street, Hanover-square, W.

1858 Turtle, Frederick, Clifton Lodge, Woodford, Essex.

1880 Tyson, William Joseph, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne-gardens, Folkestone.

1854 Vasey, Charles, 112, Cambridge-gardens, Notting-hill. W.

1867 Venning, Edgcombe, 30, Cadogan-place, S.W.

†1867 Wagstaffe, William Warwick, B.A., Purleigh, St. John's-hill, Sevenoaks. (C. 1874, 1878-80. S. 1875-7.)

O.M. Waite, Charles D., M.D., Senior Physician to the Westminster General Dispensary; 3, Old Burlington-street, W.

1885 Wakley, Thomas, jun., 96, Redcliffe-gardens, S.W.

1881 Waller, Bryan Charles, M.D., Masongill House, Cowan-bridge, Kirkby-Lonsdale.

1888 Walsham, Hugh, M.A., M.B., B.C., 134, Harley-street, W.

1873 Walsham, William Johnson, M.B., C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital, Surgeon to the Metropolitan Free Hospital; 27, Weymouth-street, Portland-place. (C. 1881-3.)

1859 Walters, John, M.B., Reigate, Surrey.

1877 Warner, Francis, M.D., Assistant Physician to, and Lecturer on Botany at, the London Hospital; 24, Harley-street, W.

1877 Waterhouse, Charles, M.B., M.C., Carl Ludwigstrasse, Währing, Vienna.

1879 Waters, John Henry, M.D., 101, Jermyn-street, St. James's, S.W.

1880 de Watteville, Armand, M.A., M.B., Physician in charge of the Electro-Therapeutical Department, St. Mary's Hospital; 30, Welbeck-street, Cavendish-square, W.

1860 Way, John, M.D., 4, Eaton-square, S.W. (C. 1873-4.)

†1858 Weber, Hermann, M.D., Physician to the German Hospital; 10, Grosvenor-street, Grosvenor-square, W. (C. 1867-70. V.-P. 1878-80.)

1876 Weir, Archibald, M.D., St. Mungho's, Great Malvern.

1864 Welch, Thomas Davies, M.D., Fairmount, Frith-hill, Godalming, Surrey.

1853 Wells, Sir Thomas Spencer, Bart., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor-street, W. (C. 1865-8. V.-P. 1876-7.)

†1851 West, Charles, M.D., 55, Harley-street, Cavendish-square, W. (C. 1856-7.)

1877 West, Samuel, M.D., Assistant Physician to St. Bartholomew's Hospital, Physician to the City of London Hospital for Diseases of the Chest, Victoria-park, and to the Royal Free Hospital; 15, Wimpole-street, Cavendish-square, W. (C. 1884-6.)

1888 Wethered, Frank J., M.B., 34, Welbeck-street, Cavendish-square, W.

Elected

1867 WHIPHAM, THOMAS TILLYER, M.B., Physician to St. George's Hospital; 11, Grosvenor-street, Grosvenor-square, W. (C. 1880-2.)

1869 WHIPPLE, JOHN H. C., M.D., Army Medical Staff.

1877 WHITE, CHARLES HAYDON, 20, Shakespeare-street, Nottingham.

1881 WHITE, WILLIAM HALE, M.D., Senior Assistant Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley-street, Cavendish-square, W. (C. 1888.)

1886 WHITE, WILLIAM HENRY, M.D., Physician to the Royal Hospital for Diseases of the Chest, 43, Weymouth-street, W.

‡1868 WHITEHEAD, WALTER, 24, St. Ann's-square, Manchester.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington-street, S.W.

1870 WICKSTEED, FRANCIS WILLIAM, Chester House, Weston-super-Mare.

1879 WILCOX, HENRY, M.B., Dorchester House, Herbert-road, Woolwich.

1869 WILKIN, JOHN F., M.D., M.C., New Beckenham, Kent.

1871 WILKINSON, J. SEBASTIAN, Late Surgeon to the Central London Ophthalmic Hospital; New Zealand.

1855 WILKS, SAMUEL, M.D., F.R.S. (TRUSTEE), Member of the Senate of the University of London, Consulting Physician to Guy's Hospital; 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-5. P. 1881-2.)

1879 WILLCOCKS, FREDERICK, M.D., Assistant Physician to Charing Cross Hospital, Physician for Out-patients to the Evelina Hospital for Sick Children; 14, Mandeville-place, Manchester-square, W.

1886 WILLETT, EDGAR WILLIAM, M.B., 60, Welbeck-street, Cavendish-square, W.

1869 WILLIAMS, ALBERT, M.D., 60, Kirkdale, Sydenham, S.E.

O.M. WILLIAMS, C. J. B., M.D., F.R.S., Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton [2, Upper Brook-street, Grosvenor-square, W.]. (*Pres.* 1846-7. V.-P. 1848-52. C. 1853-5. V.-P. 1858-61.)

‡1858 WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales-road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook-street, Grosvenor-square, W. (C. 1875-8.)

1881 WILLIAMS, DAWSON, M.D., B.S., 25, Old Burlington-street, W.

1872 WILLIAMS, JOHN, M.D., Obstetric Physician to University College Hospital; 11, Queen Anne-street, Cavendish-square, W. (C. 1878-80.)

1881 WILLIAMS, W. ROGER, Surgical Registrar to the Middlesex Hospital.

1864 WILLIAMS, W. RHYS, M.D., Commissioner in Lunacy; 19, Whitehall-place, S.W.

1876 WILLIAMSON, JAMES MANN, M.D., Ventnor, Isle of Wight.

1863 WILLIS, FRANCIS, M.D., The Spa, Bracebrough, Stamford.

1888 WILSON, CLAUDE, M.D., C.M., 6, York-road, Tunbridge Wells.

1859 WILSON, EDWARD THOMAS, M.B., Montpelier-terrace, Cheltenham.

1859 WILSON, ROBERT JAMES, 7, Warrior-square, St. Leonards-on-Sea.

Elected

‡1861 WINDSOR, THOMAS, Consulting Surgeon to the Salford Royal Hospital; Woodcroft, Dudley-road, Manchester, S.W.

1874 WISEMAN, JOHN GREAVES, Dearden-street, Ossett, Yorkshire.

1865 WITHERBY, WILLIAM H., M.D., Pitt-place, Coombe, Croydon.

1850 WOOD, JOHN, F.R.S., Surgeon to, and Professor of Clinical Surgery at, King's College Hospital; 61, Wimpole-street, W. (C. 1857-9. V.-P. 1872-4. T. 1884-7.)

1887 WOOD, T. OUTTERSON, M.D., 40, Margaret-street, Cavendish-square, W.

1854 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital; 99, Harley-street, W.

1876 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Rockhampton, Queensland.

1883 WOODCOCK, JOHN ROSTRON, 155, Hagley-road, Birmingham.

1883 WOODHEAD, GERMAN SIMS, M.D., 6, Marchhall-crescent, Edinburgh.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1887 WOOLDRIDGE, LEONARD CHARLES, M.D., 24, Trinity-square, S.E.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1869 WYMAN, W. S., M.D., Westlands, 280, Upper Richmond-road, Putney, S.W.

1884 WYNTER, WALTER ESSEX, Templecombe, Twickenham.

1869 YEO, I. BURNEY, M.D., Physician to Kings College Hospital; 44, Hertford-street, Mayfair, W.

1872 YOUNG, HENRY, M.B., Monte Video, South America.

ANNUAL REPORT OF COUNCIL, 1887–88.

PRESENTED AT THE ANNUAL MEETING, JANUARY 3RD, 1888.

THE Council has the pleasure of reporting a further increase in the number of the members of the Society during the past session, the total being now 691 as compared with 677 at the corresponding period of last year. Twenty-nine new members have been elected, of whom eight are non-resident. Three resident members have become non-resident. Six have died, namely, Dr. Andrew of Shrewsbury, Dr. Carrington, Dr. de Castro, Dr. Leudet, Dr. Wilson Fox, and Mr. Wilson Wilcox. It is impossible to pass by the names of Dr. Wilson Fox and Dr. Carrington without a word of reference to the good work done by them. Dr. Carrington at the time of his death was a member of the Council. Dr. Wilson Fox was at one time Vice-President of the Society, and in former years took a very active part in its proceedings; he made many very valuable communications to its 'Transactions,' and in 1873 he opened a discussion on the anatomical relations of pulmonary phthisis to tubercle, which, it will be remembered, was the first of a series of discussions on special subjects, which have added largely to the usefulness of the work of the Society.

The balance at the Society's bankers is much smaller than that of last year, although the income has been somewhat larger, viz. £593 3s. 0d. as compared with £583 14s. 1d. This is due to the fact that the Council have thought it wise to devote a certain amount of the Society's funds to the compilation of a general index

of the contents of vols. xxvi to xxxvii. The present seemed the most suitable time for this undertaking because in twelve years the fiftieth volume of the 'Transactions' will have been published, and it is probable that a complete general index up to date will be made. The present is the third of such general indexes, the first of which included the contents of vols. i to xv, and the second those of vols. xvi to xxv. The cost of its production has been £156 3s. 10d., and the Society is to be congratulated on the fact that notwithstanding this large outlay it has not been found necessary to draw at all upon its funded capital. In this connection it may be pointed out that certain alterations in the type of the index of the volume have been made this year which it is hoped will render it more clear and more suitable for reference than heretofore.

Some modifications in the manner of describing card specimens have been introduced by the Council with the object of making more valuable the records of those which are published in full in the 'Transactions,' whilst those of minor importance are to be simply catalogued. The method in which the card specimens are described will, however, be left to the discretion of the exhibitors.

Beyond these points there are no others of importance to record in the management of the Society during the past session, which has, in the opinion of the Council, been one of continued prosperity and activity.

James Paget.

	£ s. d.	£ s. d.
By *Balance* at Bank, January 1st, 1887		154 1 8
Subscriptions:		
410 Annual (Current)	430 10 0	
5 Ditto (in Advance)	5 5 0	
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		471 9 0
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January, 1887	16 18 7	
July, 1887	17 0 1	
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		£747 4 8

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Microscopes and Lamps	16 4 0	
Chairs (President's Address)	1 12 0	
Morbid Growths' Committee (Assistance)	1 0 0	
		141 16 0
Transactions: Vol. XXXVIII:		
Printing, Binding, Delivering (Adlard)	211 18 7	
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Ditto (Mr. Sutton)	1 10 0	
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		342 9 10
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Ditto (Medical Secretary's)	1 6 10	
Commission on Collecting Subscriptions	18 12 0	
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New General Index to Vols. XXVI—XXXVII:		
Compiling by Mr. Bailey	50 8 0	
Printing, Binding, &c. (Adlard)	105 15 10	
		156 3 10
Balance at Bankers		38 11 0
		£747 4 8

January 3rd, 1888.

Audited and found correct, W. B. HADDEN, D'ARCY POWER, } *Auditors.*

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REPORT.

SESSION 1887-1888.

I. DISEASES, ETC., OF THE NERVOUS SYSTEM.

1. *Partial perforation of skull in a case of cerebral tumour.*

By J. A. Ormerod, M.D.

The skull-cap shows in the right frontal region the upper part of a large opening somewhat square in shape. The greater part of this opening was made in the course of a surgical operation, but there existed beforehand a small though complete perforation here, caused by the cerebral tumour. In the rest of the skull-cap are several holes, about thirty in number, rounded in shape, size from that of a large pin's head to a quarter of an inch in diameter, with edges clean cut and not thickened, in depth extending through the inner table, and sometimes almost through the outer table, so that here their base is covered in by an extremely thin layer of bone. These holes are scattered over the frontal and parietal bones, the largest being on the left side.

In addition there is some general thickening of the skull, at the upper part of the parietal bones, and in the neighbourhood of the large (surgical) perforation.

Further, the bone between the holes is remarkably roughened, the inner table being studded with little eminences, which have sharp and almost thorny apices.

In the dura mater are numerous clean-cut perforations answering to the holes in the skull.

When the skull-cap was removed, it was seen that the perforations in the skull corresponded to small outgrowths from the

brain, which sprouted through the dura mater, looking like small Pacchionian bodies in the wrong place. They were soft, and of the colour of brain-tissue, and grew from the brain-substance, with which they seemed to the naked eye continuous. Into some of these the white matter appeared to pass up, like the vessels into an intestinal villus.

The conditions in the interior of the brain itself may be shortly stated thus. There was a large slough at the anterior extremity of the right frontal lobe, the site of the surgical operation, which filled up the opening there made in the skull during life. Behind this lay a large tract of white soft matter which may have been either tumour or softened brain. The outlines of the cortex were here indistinguishable. This soft tissue reached backwards to the roots of the horizontal frontal convolutions, and in depth involved the whole thickness of the frontal lobe. The basic ganglia were softened on the right side, but not on the left. In the ventricles was a dirty-looking fluid; the choroid was infiltrated with pus. This suppurative process, beginning probably at the right frontal lobe, had extended through the ventricles down into the descending cornu on the left side. There it had caused a large abscess in the temporo-sphenoidal lobe, which had almost perforated at the tip of the lobe. There was some (secondary) superficial necrosis of the bone here, which would seem to show that the abscess was not of recent formation. There were no tumours elsewhere in the body.

Brief abstract of clinical history.—The patient, a police constable, aged 30, was admitted into St. Bartholomew's Hospital, under the care of Dr. Andrew, on September 25th, 1886.

His previous history was unimportant. He had had frontal headache, principally right-sided, for six months, vomiting, and loss of memory; difficulties with his bladder for six weeks; and some weakness of the legs, twelve months.

On admission, he could only just stand; his urine was passed under him; he had to be fed. There was double optic neuritis. The superficial reflexes and tendon reactions were normal. There were no localising symptoms.

Subsequent observations as follows:

October 18th.—Slight ptosis (left) and (later) contracture of left arm. Foot-clonus, both sides. Patellar tendon reaction absent right side.

During the rest of the year gradually increasing drowsiness; he had to be fed by the nasal tube. The optic discs became atrophic.

By April, 1887, he was quite comatose, but there still remained some distinction between sleeping and waking, and he resisted being fed.

In September, 1887, a small ulcer on right cornea. The head seemed growing longer, the frontal region is described as falling in laterally.

In October, 1887, a fluctuating swelling appeared over the right eye; the skin was incised, and a probe could be passed into the interior of the skull.

November 1st, 1887.—The swelling was laid open by Mr. Marsh, and a small round opening was found in the skull, from which a soft reddish mass protruded. After removal of a considerable piece of bone, a large soft mass was exposed to view, which could not be delimited from the brain. Much of it was scooped away, but it went too far back to be entirely removed. A cheesy mass was found in its centre. There was considerable hæmorrhage, for which the actual cautery had to be used. No improvement was obtained in the general condition. The wound became foul, and a large hernia cerebri formed. Death on November 8th, 1887.

It appears therefore that the general symptoms of cerebral tumour were present on admission, but none by which the disease could be localised. A little later left-sided paralysis appeared. A year later, and a few weeks only before death, actual perforation occurred above the right eye, probably by the same process as the perforations which are now shown on the vertex.

It should be added that in spite of the coma, the inability to swallow, and the involuntary passage of excreta, this patient was kept alive and without a bedsore for over a year.

Mr. Bowlby examined the tumour when removed by operation, and found it to be a sarcoma. *December 20th*, 1887.

2. *Angioma of the cerebral membranes.* (*Card specimen.*)

By D'Arcy Power, M.B.

[With Plate I, fig. 1.]

The angioma forms an oval swelling situated upon a vein on the cerebral surface of the pia mater covering the angular gyrus. The tumour itself, after preservation in spirit, measures an inch in length and three quarters of an inch in diameter. It consists of a close plexus of vessels, which have an average diameter of an eighth of an inch, and are therefore of sufficient size to receive a coarse injection of gelatine and carmine. The vessels appear to be derived from the vein upon which they lie, reinforced by branches from the neighbouring veins.

From a young man aged 20, who suddenly became comatose after returning from work in an apparently healthy condition. Whilst he was comatose he had left hemiplegia with divergent strabismus. Two days after the seizure the patient had several attacks of opisthotonos with spasm of the left limbs. He died ten days after the onset of the symptoms.

At the *post-mortem* examination a large irregular hæmorrhage was found in the right cerebral hemisphere. The blood appeared to come from the vessels immediately surrounding a tumour which was situated upon the surface of the right angular gyrus, though its exact source was not determined. The hæmorrhage extended from the surface of the brain to the posterior part of the internal capsule, and had neither burst into the lateral ventricle nor externally. The spinal cord and other organs of the body were quite healthy.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series xxx, No. 2466(a). *February 7th*, 1888.

DESCRIPTION OF PLATE I.

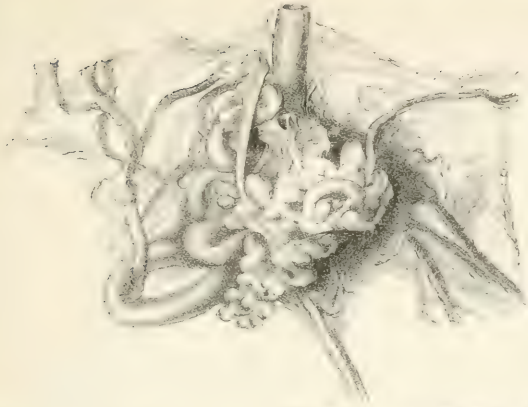
Fig. 1.—To illustrate Mr. D'Arcy Power's specimen of Angioma of the Cerebral Membranes. (Page 4.)

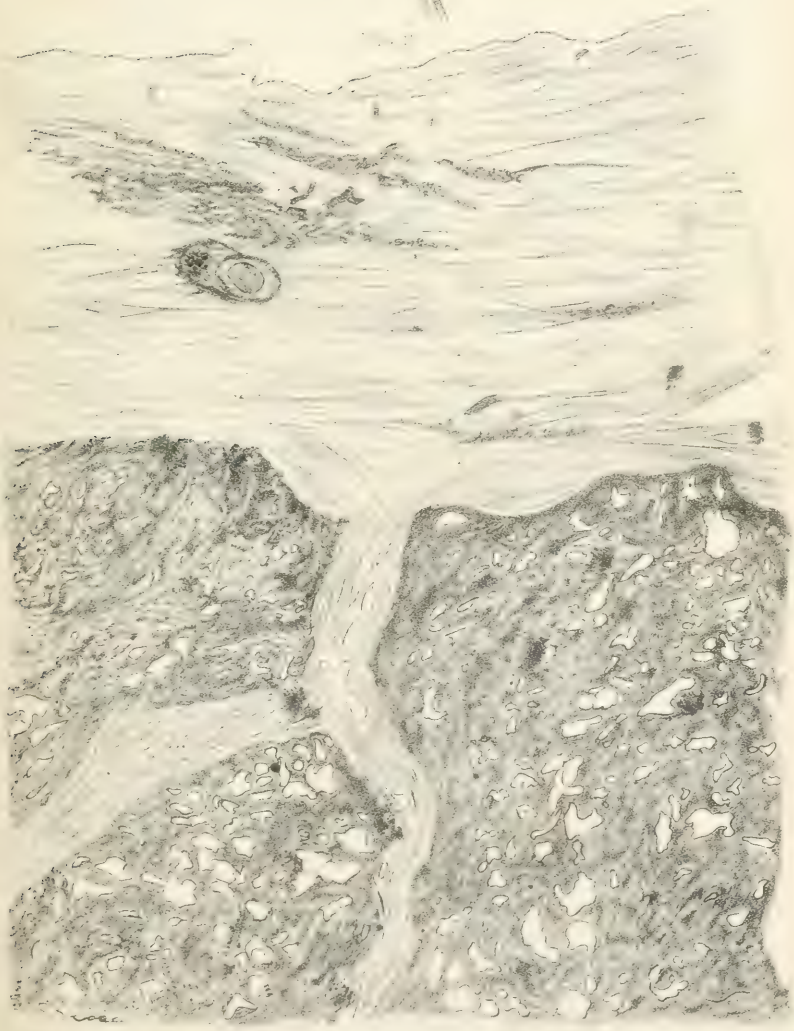
Fig. 2.—To illustrate Dr. Handford's case of Carcinoma of Lung. (Page 53.)

From a drawing by Mr. James Elliott.

Carcinoma originating in the mediastinum and involving the lung. A process of the greatly thickened carcinomatous pleura spreading into the lung, which is extensively consolidated, partly by pneumonia, but chiefly by carcinoma (epithelial cells and small-celled infiltration). Obj. 2 inches, oc. 2.

Fig. 1.





3. *A case of primary cancer (or endothelioma) of the brain, in connection with peduncles and pons.*

By Joseph Coats, M.D.

[With Plate II.]

I have been induced to bring forward this case chiefly on account of its unusual character, and because it raises questions as to the origin of cancer which still await answers. It was somewhat of a surprise to me to find a tumour, apparently growing from the deeper parts of the brain, composed of a tissue in many ways analogous in structure to an epithelioma of the alimentary canal.

During life the patient was for some time a patient of Prof. Gairdner in the Western Infirmary, Glasgow, and he has been good enough to furnish me with the following notes of the clinical history.

Mrs. G—, aged 32, was admitted to the Western Infirmary (Ward 6) on July 18th, 1887. Her complaint seemed to have taken origin after a confinement in November, 1886, when she became subject to "weak turns," which, as described, seemed at times to have involved loss of consciousness, but were very imperfectly described. She had also occasional vomiting after these attacks. Six weeks before admission pain in the head began, accompanied sometimes by vomiting and retraction of the head. This pain became extreme, causing her to cry out as in agony, and also to talk nonsense. There was also a degree of deafness.

On admission, a peculiar expression was noted, arising from a degree of ptosis, with a little external deviation of both eyes. There was engorgement of the optic nerves, engorgement and tortuosity of the small vessels extending for some distance beyond the discs, with traces of exudation over the discs and beyond them along the course of the vessels. The temperature, on one occasion only, reached 101·6°, but, with comparatively few exceptions, was normal throughout. A month after admission it was noticed that the patient had been gradually becoming more lethargic, so as to be with difficulty aroused, her answers even when aroused being very stupid, and only after repeated and loud questioning. She

was apt also to rise from bed aimlessly and to utter incoherent remarks. She complained constantly of the pain in her eyes, the vision having been more or less defective even from the commencement of the symptoms. A slight degree of left facial paralysis was observed on one occasion, but no paralysis of limbs.

In the latter part of August she began to pass her evacuations in bed, and was still more lethargic and incoherent. From this time she seemed to lose ground from day to day, becoming much emaciated, though still taking nourishment, and without any new cerebral symptoms. Breathing quiet and regular, pulse regular, 80 per minute.

On September 19th, a quite exceptional rise of temperature took place to 102°, associated with a rapidity of pulse never before observed, 140, and some complaint of pain in the right side. There were râles also at right apex. No considerable change took place in the cerebral condition, but the patient on the whole was manifestly worse, and continued in a similar state till the evening, when she died.

Post-mortem appearances.—On removing the brain and examining it from above, a prominent grey tumour was observed, presenting itself between the cerebellum and the cerebrum in the middle line. The tumour appeared in its anterior parts to be cystic or softened, only a thin layer of tissue confining the soft contents. After hardening in alcohol a longitudinal section was made in the middle line so as to show the relations of tumour and neighbouring structures. This section is represented in Plate II, fig. 1.

The tumour was seen to lie above the pons Varolii and the peduncles, and though generally in the middle line, it had more of its substance to the right than to the left. It impinged on the pons, but seemed to be more immediately in connection with the peduncles, projecting upwards and backwards from these.

Its relation to the aqueduct of Sylvius and fourth ventricle is interesting, and is such that it may have originated from the aqueduct. The first part of this passage, for the distance of a quarter of an inch, is in front of the tumour, and is not appreciably affected. The remainder of it is flattened over the convexity of the tumour, which may be said to dilate this portion of the aqueduct. The structures above the aqueduct are greatly compressed and elongated backwards, the corpora quadrigemina being only distinguishable as an elongated arched layer of nervous tissue about a

DESCRIPTION OF PLATE II.

Illustrating Dr. Joseph Coats' case of Primary Cancer of the Brain. (Page 5.)

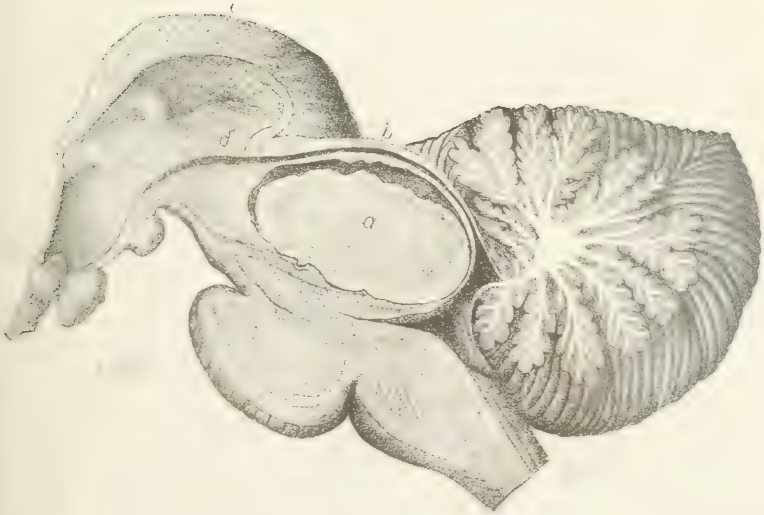
From drawings by Dr. Coats.

FIG. 1.—Median section exposing tumour and neighbouring structures of brain. *a*. Tumour. *b*. Corpora quadrigemina stretched over tumour. The aqueduct of Sylvius is visible between tumour and corpora quadrigemina. The tumour projects backwards into fourth ventricle. *c*. Thalamus opticus. *d*. Third ventricle.

FIG. 2.—From marginal part of tumour. Along upper surface it is in immediate contact with brain-substance (*a*), laterally it is in contact with connective tissue, which is somewhat irritated. × 70.

FIG. 3.—An isolated part in midst of tumour. It encloses connective tissue in which are the open mouths of a number of vessels. × 70.

Fig 3



sixteenth of an inch in thickness. The calibre of the aqueduct can still be made out between the tumour and the corpora quadrigemina. The tumour projects posteriorly into the fourth ventricle, a considerable portion of its bulk being in this ventricle.

The tumour itself is oval in shape, the long diameter, which is from before backwards, being an inch and three eighths in length, and the short diameter, which is from above downwards, about seven eighths. The posterior part of the tumour is solid, and consists of a soft whitish tissue, which towards the central parts is obviously brittle, while completely softened and crumbling anteriorly.

There was a moderate degree of hydrocephalus, the surface being somewhat glazed and the ventricles containing an excess of fluid, of which it was possible to collect about an ounce.

Microscopic examination.—The tissue of the tumour is evidently very soft, and continuous sections are only obtained by embedding in celloidin. In sections so obtained the tissue is seen to be frequently broken up, even at the marginal parts of the tumour.

The most striking constituents of the tumour are cylindrical cells arranged in rows or enclosing spaces. This is very strikingly seen at the margin of the tumour, where it impinges on the brain-substance. Here there is an apparently continuous row of palisade-like cells, which form a sinuous or convoluted outline (see Plate II. fig. 2). These cells almost directly impinge on the brain-substance, there being at most an irritative layer infiltrated with round cells between them and the proper nervous tissue. The cells are in the form of typical cylindrical epithelium, and strongly suggest those which line the ventricles or central canal of the cord. Throughout the tumour these cylindrical cells are present, sometimes enclosing spaces, and sometimes forming more elongated sinuous rows.

The relation of these cylindrical cells to the remaining constituents is somewhat peculiar. The cylinder cells, lying close together, have flattened summits, which form a level continuous surface, like the flat surface of the cylindrical epithelium of any mucous membrane or of the epithelium of the central canal. This appearance would suggest that the epithelium was lining glands or ducts, but this flat surface is in contact with connective tissue, which is usually very vascular. In fact, just as at the margin of the tumour the cylindrical cells face the nervous tissue, and are here in contact with vascular structures, sometimes infiltrated with

round cells, so here the cylinders are in contact with connective tissue, and the spaces which the rows of cells enclose are occupied by highly vascular connective tissue (see fig. 3.) On the other side of the cylindrical cells there are irregular aggregations of large cells having the characters of epithelial cells. Sometimes they form large globular bodies as if swollen up, and this is associated with obscuration of the nuclei. These cells are of various shapes and sizes, and they are apparently very prone to secondary changes.

It should be added that the cylinder cells are obviously adherent to each other, so that they often, in the broken parts of the tumour, are seen in short lengths, a number lying stuck together and separated from the rest. No cilia were detected on the cells.

It must be confessed that this tumour presents in its structure certain differences from an ordinary cancer. The cylindrical cells do not in any sense line ducts, or even form the regular lining of cell-nests. On the other hand, their typical epithelial character almost compels us to place the tumour among the epithelial tumours, and to call it a cylinder-celled epithelioma. I think that in this case the tumour has probably originated from the epithelium lining the aqueduct of Sylvius, and if we regard this as epithelium and not as endothelium, then again, in reference to its origin, the tumour should be called an epithelioma. I am aware that many pathologists deny the possibility of a primary cancer of the brain, and would call such a tumour as this an endothelioma, but I cannot say that I am convinced of the correctness of their views. *May 1st*, 1888.

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4. *Psammoma of brain.* (*Card specimen.*)

By L. A. Dunn, B.S.

This specimen of cerebral tumour was removed from the brain of a subject in the dissecting room at Guy's Hospital. It was firmly attached to the dura mater and also to the inner aspect of the right parietal bone, the point of its bony attachment being situate about three quarters of an inch external to the centre of

the upper border of the bone and marked by a rough eminence, pitted in places.

The tumour appeared to occupy the right parietal lobe, but such was the diffluent condition of the brain that an exact observation could not be made. The tumour is of an irregular, spheroidal shape (two and a half by two inches), firm on section, and fibrous looking. By the aid of the microscope it is found to consist of fibrous tissue mainly, with here and there "nests" typical of a psammoma. On applying to the medical officer of the infirmary at which the patient died I was informed that death was due to bronchitis and that no symptoms of cerebral compression whatever were evinced by the patient during life. *May* 1*st*, 1888.

5. *A case of cystic growths in the cerebellum and right adrenal.*

By F. Charlewood Turner, M.D.

The two preparations exhibited show the cerebellum with the pons Varolii and medulla oblongata, and the right adrenal of a man aged 44, who died with symptoms of tumour in the cerebellum.

In that organ there is, at the outer border and under surface of the right lobe, a tumour about the size of a pigeon's egg, consisting of a cyst surrounded by a wall of new growth a quarter of an inch thick, except on its under surface, where it was closed in by membrane only. The cyst was lined by a smooth membrane and contained clear serous fluid. This tumour, though really structurally connected with the cerebellar cortex, appeared to be completely encapsuled and attached to the nerve-structures by membrane only.

At the upper surface of the cerebellum there is a second smaller tumour, of rounded form and about one inch in diameter, which projects at the inner part of the antero-superior lobe on the same side, in immediate contact with the middle lobe at that part, which it had compressed and indented on the right side. This tumour has been laid open in its longest diameter by an obliquely transverse incision. It is seen to be separated by a capsule from the contiguous laminæ of the cerebellum, which it has displaced, but

to be in structural continuity with the medullary tissue at its deep surface. Over its projecting surface the flattened remains of atrophic laminæ pass from side to side. In the central and upper (or outer) part of this growth is a cavity of the size of a broad bean, filled with granular coagulum of light brownish tinge. In both tumours the growth has a brownish colour mottled with darker spots, indicating its great vascularity and the occurrence of hæmorrhages in it.

The right adrenal has been converted into a tumour of the size of a fist, and consists of a large cavity divided into two by a rigid longitudinal septum, which looks like a thin plate of cartilage, enclosed by a wall of new growth from a quarter to over half an inch in thickness. The growth is in parts fibrous, and in places there are opaque necrotic areas. There are numerous cysts in the section from the size of a split pea and less. The central cavity has a thick fibrous wall which is covered by masses of opaque granular coagulum. It contained a quantity of gelatinous matter and fibrinous coagulum when laid open at the autopsy. The tumour is covered on one side by peritoneum.

The following is an abstract of the clinical notes of the case :

George P—, aged 44, described as a labourer and a temperate man, was admitted into the London Hospital under the care of Dr. Down on January 20th, 1887, complaining of violent headache, which had lasted eighteen months, and of sickness, which had come on lately. He had served in the Police Force ten years, getting knocked about a good deal. Eight years ago he was hit on the forehead by a brick. Twenty years ago he had "typhus" fever, and, about eight years ago, ague. There was no history of gout or syphilis. He himself attributed his illness to want of rest and anxiety. Eighteen months ago, when at work, he was attacked with pain in his head. This continued more or less from that time, but about a week before Christmas it became so severe that he had to give up work, and it has been nearly constant since then.

It was most severe in the frontal and occipital regions but also at the vertex. Since Christmas he has had attacks of vomiting, especially in the morning and evening, about half an hour after food. He suffered also from buzzing in the ear, deafness, and dimness of sight.

When admitted, he was an intelligent man, well built, and fairly well nourished, but weakened by his illness, depressed in spirits,

and emotional. He spoke slowly, and sometimes scarcely audibly, and could only walk for a few yards, getting giddy and staggering, with pain in his head. He was not in pain and fairly comfortable when lying quiet. He slept badly. There was no muscular paralysis or tremor, but he could not write so well as before. No defect of tactile sensibility but a sense of numbness over the head. The knee-jerks and plantar reflexes were present. There was no notable evidence of visceral disease except some tortuosity of the arteries.

At a subsequent examination it was found that he had a tendency to fall towards the right side when he stood up or sat up on the side of the bed. There was marked inco-ordination in walking.

On ophthalmoscopic examination (by Dr. James Anderson) the right optic disc was found to be swollen and its edges blurred, and numerous small hæmorrhages were seen. In the left fundus no hæmorrhages were visible, but the inner half of the disc was of a full pink colour, and the vessels were hazy, the outer half of the disc being pale. The movements of the eyes were natural though somewhat slow, and there was no inequality of the pupils, which reacted to light and accommodation, though feebly. He could not hear a watch within an inch of his right ear, or in contact with his right temple, but he could hear it well when placed against his left temple or six inches from his left ear. The tongue deviated to the right when protruded. There was no facial paralysis.

After admission headache, varying in intensity, and vomiting, with constipation, continued the most prominent symptoms. Later on, increasing enfeeblement of the muscular movement was very notable. The patient became drowsy and apathetic, passing the evacuations in bed, and ultimately died comatose with pneumonia of the right lung. His temperature was subnormal, ranging from 97° to 98·4°, until a few days before his death. The urine was natural.

Autopsy.—At the autopsy inspection of the brain revealed the cerebellar growth above described, and dilatation of the lateral ventricles with flattening of the convolutions of the hemispheres. The spinal cord was normal. The left adrenal was normal. In the liver there was a cyst of the size of an almond, about the middle of the upper surface, filled with clear fluid, in which were suspended small plates of cholesterine, about one sixteenth of an inch square and less than half that thickness; and in the gall-bladder,

in which there was an annular constriction about three quarters of an inch from the fundus, were several pale fawn-coloured cholesterine calculi, of rounded shape, partially facetted. In the left kidney were several cysts both superficially and deeply situated. There was pneumonic consolidation of the upper part of the right lung. The heart was normal with the left ventricle contracted. There was no enlargement of the glands along the spinal column. No bronzing of the skin.

Microscopical examination of the growth.—Thin sections were made from the growth in the wall of the cyst in the larger tumour of the cerebellum and from the adrenal growth, and are exhibited with the specimen. The former was found to consist of a highly vascular growth with many greatly dilated vessels, some filled with blood-coagulum, some empty or with shreds of coagula adherent to their walls. These varicosities of the vessels occupy much of the field. The growth itself consists of a fine reticulum, indistinct and cloudy in the sections, in which are round and elongated oval nuclei of considerable size but faintly stained, and smaller deeply stained leucocytes and elongated nuclei in the walls of the vessels. The nuclei are not very abundant and nowhere crowded together. In places the structure has a cloudy, fibrous appearance without nuclei. The cyst wall is well defined and fibrous. On one side of the section, where the free surface of the growth has been included, atrophic remains of the granular and superficial layers of the cerebellar cortex are seen, with some of the cells of Purkinje, showing that the growth was in the medullary matter of the cerebellum, though apparently completely encapsulated and separated from it.

The sections from the adrenal growth exhibit a similar but more fibrous, sparsely nucleated, reticular structure, traversed by a network of dilated capillaries with thickened walls, and here and there by supporting columns of dense fibrous tissue (lymphadenoid tissue). There are also many large vessels conspicuous in the section; and the larger spaces, which give the growth a cystic appearance, seem to be large vascular channels in it. In parts the tissue is degenerated, consisting of a cloudy, fibrous meshwork traversed by vessels.

At the outer surface of the growth, beneath its thick fibrous capsule, there is a band of different structure, separated from the lymphadenoid structure above described by a broad tract of dense fibrous tissue. This comparatively narrow band, which seems to be

the altered cortical tissue of the adrenal, consists of a closely felted fibrous structure, like that seen in advanced conditions of cirrhosis of the kidney, traversed by a network of vessels of varying calibre, so that its section has the appearance of a perforated membrane. The glandular structure of the organ has entirely disappeared.

Remarks.—The clinical symptoms in this case accord with those generally associated with tumours involving or compressing the middle lobe of the cerebellum, as illustrated in the case of tumours in this situation in the series of cases of cerebral tumours exhibited to the Society last session.

In two of these cases the symptoms were associated with injuries to the head. In one instance a sarcomatous tumour affecting chiefly the central and left lobes of the cerebellum was attended by symptoms dating from six months after an injury to the forehead on the right side. It seems possible that the growths in the present instance may have been in part dependent upon injury sustained by the cerebellum through *contre-coup* from the blow on the forehead from a brick, although said to have occurred six years before the commencement of the symptoms, the eighteen months' duration of the early symptoms of headache, showing the slowness of the growth.

The cerebellar tumours may, however, have been secondary to the larger and more fibrous abdominal growth, being of a very similar structure, and multiple primary growths in the brain being especially rare.

The concurrence of "simple cysts" in all these growths, with cysts in the liver and kidney, may have been an accidental association of cysts of different kinds, connective-tissue cysts in the tumours and glandular in the liver and kidney. But the fact is of interest in connection with cases in which cysts in various organs have been found associated, such as those recorded by Drs. Savage and Hale White in vol. xxxv of the 'Transactions,' and especially one recorded by Dr. Pye-Smith in vol. xxxvi of the 'Transactions,' in which there were numerous small cysts in the pancreas and kidneys with a simple cyst in the cerebellum.

November 15*th*, 1887.

6. *A case of necrosis of mastoid cells; trephining; spinal meningitis; death.* (*Card specimen.*)

By John R. Lunn.

J. D—, aged 20, male, November 26th, was admitted suffering from otorrhœa. Shortly before admission the discharge stopped. This was followed by vomiting, headache, opisthotonos and dilatation of the pupils and high temperature. These symptoms continued till on the 8th of January he was trephined; some fetid pus and dead bone came away. All symptoms ceased, the wound was drained and healed kindly, and patient seemed to get quite well.

On February 1st, the symptoms began to return. Vomiting first appeared, followed by high temperature, opisthotonos, weak rapid pulse, pains in limbs, and sluggish pupils. These symptoms continued till his death on February 16th.

On examining the cord it was discovered that from the medulla down to the cauda equina was a thick layer of greenish pus under the arachnoid, thicker in the lumbar than cervical regions. The cord itself was congested and softened. The base of the brain showed all signs of inflammation of its membranes, especially around the pons and cerebellum. Brain and cerebellum healthy.

February 21*st*, 1888.

7. *Syringomyelia, with dilatation of the lateral ventricles of the brain.*

By F. Charlewood Turner, M.D.

The specimen exhibited is the spinal cord of a woman aged 23, who died in the London Hospital from a severe burn, the result of her having fallen into the fire in a fit. She had had wasting of the arms during twelve months, the limbs presenting the appearance characteristic of progressive muscular atrophy.

She had been subject to fits for several years, and had three or

four during the night after her admission, but none afterwards. She had pyrexia of moderate degree while in the hospital (100°—101°). She had persistent diarrhœa for three days before her death, which occurred on the thirteenth day after admission.

The spinal cord is traversed throughout the cervical and dorsal regions by a large, centrally situated tubular cavity of irregular form, and in the lower half of the lumbar enlargement and conus by a smaller cavity in the right posterior grey column, unconnected with the other.

The larger cavity occupies both halves of the cords, and is centrally situated excepting at its upper extremity in the lower part of the medulla oblongata, where it appears in sections through the decussation of the pyramids, as a transverse fissure in the continuation of the left posterior cornu, nearly reaching the surface of the cord, and one in the substance of Rolando on the right, and in the middle of the dorsal region, where it is divided into two parallel channels in the posterior part of the grey substance on each side. Where centrally situated, it is divided by an incomplete median antero-posterior membrane.

In the *cervical* region the transverse section of the channel is rounded anteriorly, but projects posteriorly along the nerve-roots to the surface of the cord, separating the posterior and lateral white columns from each other. Microscopical examination of thin sections in this and in the dorsal regions shows that the cavity has been formed in the posterior part of the grey columns and commissure, lying behind the central canal. In the middle and upper part of the cervical enlargement, where the expansion of the cord is greatest, the section is nearly symmetrical; above and below, the expansion is greater on the left side.

At the junction of the *dorsal* and cervical regions the channel is smaller, contracted and rounded on the left side by disappearance of the projection of the cavity along the posterior nerve-roots on that side. At the same time the section of the channel is enlarged on the right side by the appearance of a rounded projection in the position of the anterior cornu and nerve-roots on that side, which very nearly reaches the surface of the cord. By this change the channel is brought more and more to the right side, and in the upper third of the dorsal region, and towards the middle, it is almost entirely on that side. It then becomes greatly contracted, and is divided into two separate and unequal channels for a short

distance in the mid-dorsal region. The two channels are in the posterior part of the grey columns on each side. That on the left is of small size, and deeply situated in the posterior cornu, that on the right is of larger size, and of triangular section, with projections in the direction of the anterior and posterior cornu and nerve-roots, and towards the central grey commissure. A little lower down a single channel is again formed, at first symmetrical with projections along all four cornua and nerve-roots, then much contracted and of triangular form almost wholly on the right side, and lastly, at the lower end of the dorsal region, once more symmetrical, rounded anteriorly and projecting along the posterior nerve-roots to the surface of the cord. Here it contracts, at first gradually and then abruptly, its lower extremity appearing in sections though the upper part of the lumbar enlargement as a narrow fissure in the inner part of each posterior cornu extended along the issuing nerve-roots, and running parallel to their border. The fissure on the left side is the larger, and that alone appears in sections a little lower down. Each fissure at its extremity is limited to the tip of the cornu and the nerve-root.

Transverse sections through the middle of the lumbar enlargement show a cavity in the outer part of the posterior cornu of the right side. This cavity is of triangular section with processes in the direction of the nerve-roots and towards the centre of the grey commissure. This second cavity is larger in sections through the extremity of the lumbar enlargement and conus, but limited to the same part. A small glass rod passed through it in the end of the conus is seen emerging upon the posterior surface of the extremity of the cord.

Sections of the cord from different regions showed that the cavity was, in this case as in others, independent of the central canal and situated behind it. In the lower lumbar region the central canal presents a normal appearance with a well-defined lining of columnar epithelium. In other parts the central canal is closed by a corpuscular mass in which its outline is obscured.

In the brain both lateral ventricles were dilated. The left anterior cornu was more expanded than the right, and the left posterior cornu extended farther back than the right.

There was nothing noteworthy in the abdominal or thoracic viscera. There were no ulcers in the duodenum.

The association of dilatation of the ventricles of the brain with

such tubular cavities in the cord, observed in similar cases generally, is emphasised in this instance in the association of a spinal lesion affecting more especially the right side of the cord with a greater ventricular dilatation on the opposite side of the brain. The facts indicate the origination of both spinal and cerebral lesion in some structural or nutritional defects affecting the cerebro-spinal system as a whole.

Leyden regards these tubular cavities in the cord as due to the distension of unobliterated portions of the posterior projection of central canal of the embryo; and Dr. Taylor mentions this as the most probable explanation of the lesion in two cases observed by him and recorded in vols. xxix and xxxv of our 'Transactions,' the latter of which resembled this specimen in several points. In that case also the cavity was at one part divided into two channels in the lateral grey columns, and in part limited to the right posterior grey cornu, being reduced to a narrow fissure in this situation in the lumbar region, and the cavity was more expanded on the left side at its upper end.

But the anatomical features of the lesion in both these specimens seem to me opposed to that view of its origin. In the present instance the limitation of the smaller cavity in the lumbar enlargement to the posterior cornu of one side, the similar limitation of the large cavity in part of its course to the posterior part of the grey column on each side, and the presence of an antero-posterior median septum in the cavity, where centrally situated, in both the cervical and dorsal regions, seem to indicate the origination of both cavities in the posterior cornua, with extension to the commissure in the case of the larger one.

The occurrence of progressive amyotrophic paralysis in the arms during the last twelve months of the patient's life, is evidence of progressive expansion of the larger cavity by increased accumulation of fluid in it. And the anatomical features of the specimen show that this accumulation of fluid by which the symptoms were produced, took place not in one central channel but in parallel channels, one in each half of the cord, in part separated by a tract of nerve-tissue, for the most part by an imperfect membranous septum only.

Thus if the lesion *originated*, in accordance with Leyden's view, in a cyst in the commissure behind the central canal, the symptoms must have resulted from an extension of the central cyst to the

posterior grey cornua, and upwards and downwards in the less resistant structure thus afforded for its expansion. The smaller cavity in the lumbar enlargement must then also have resulted from the downward extension in the right grey column of the larger cavity from which it appears to be completely separated.

It is, I think, more probable that the cavities originated as simple connective-tissue cysts, such as those seen in the cerebellum or in the membranes of the brain, in the posterior grey columns on both sides of the spinal cord, these being the parts in which, as the specimen shows, the resistance to the accumulation of pent-up cerebro-spinal fluid or serous exudation is least; both sides being simultaneously affected as in other independent and intrinsic lesions of the cord.

The association of the spinal lesions with distension of the ventricles of the brain is also more in accordance with this view of the origin of these cavities. For the ventricles of the brain, though developmentally and anatomically connected with the central canal of the spinal cord, are, as large reservoirs for the cerebro-spinal fluid and quasi-perivascular spaces for the vessels of the velum interpositum, more in correspondence physiologically or functionally with the perivascular and other lymph-spaces of the connective tissue of the cord. *December 6th*, 1887.

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8. *Case of Syringomyelia.*

By A. Quarry Silcock, M.D.

[With Plate III.]

The microscopical specimens and drawings are shown with the purpose of demonstrating the formation of a tube-like cavity, occupying the posterior columns of the cord and extending through a considerable length of the latter, by a process of excavation of newly-formed connective tissue which has developed in that situation as a result of traumatic myelitis.

The cord was taken from the body of a man who broke his neck

in diving into the river Thames. The autopsy, and a subsequent examination of the cervical spine, showed that the body of the fifth cervical vertebra had been crushed and somewhat displaced backwards, giving rise to a projection in the spinal canal. Opposite this projection the spinal cord was almost completely disorganised, evidently by the pressure exerted upon it. The clinical history of the case is remarkable, insomuch as the man lived exactly ten months after the accident. It is fully set forth in the 'Lancet' of October 1st, 1887, by Surgeon-Major Myers, who kindly sent me the cervical spine and cord for examination.

The following is a brief excerpt from Surgeon-Major Myers' account of the case:—

"Private S—, Grenadier Guards, a very strong man, aged 23, weight 14 st., was admitted to the Windsor Station Hospital on July 3rd, 1886. He dived head foremost into shallow water (two feet), and was taken out almost immediately by some comrades, who noticed that his legs were completely paralysed, and he was at once conveyed to the hospital. When seen shortly afterwards, he was lying on his back, perfectly conscious, but devoid of sensation and motion in the lower part of the trunk and legs. Slight sensation was present to about the lower angle of the scapulæ, and there was some power of movement in both arms at the shoulder; respiration was entirely diaphragmatic; pulse slow and strong. The patient complained of no pain excepting at the back of the neck in the region of the fifth and sixth cervical vertebræ, and chiefly on the left side.

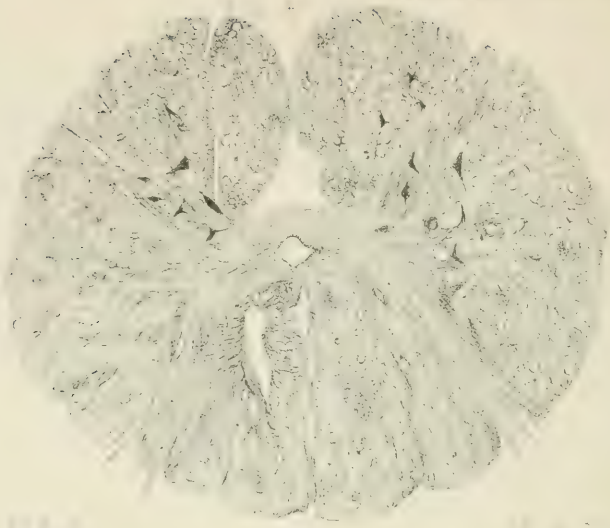
Some days after admission the arms became motionless, excepting a slight power to raise the elbow from the side, and remained flexed. The sensation returned in the right arm, but it soon became apparent in the left arm as low as the elbow, and extending over the anterior surface of the radial half of the forearm, and at a later period in the tips of the thumb and first and second fingers. Over and above the shoulders there was marked hyperæsthesia. For many weeks bedsores were well kept in check, though continually threatening, but in course of time they formed in numerous places, especially along the under surface of the right leg, and even on the toes, where no pressure occurred. At a later stage the integument of the back became completely undermined and converted into a large sac, containing very offensive pus. This sac, by irrigation twice daily with disinfectants through the sacral bedsore,

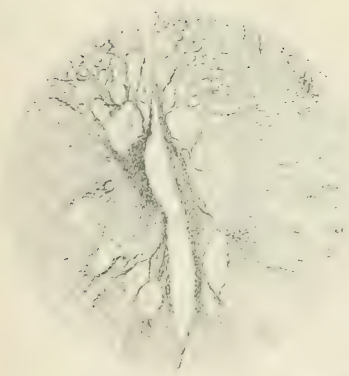
completely disappeared; and a short time prior to death the bedsores of the right leg had healed, and others over the scapulæ, elbows, and hips had nearly done so, whilst even the deep sacral bedsore had greatly contracted. Cystitis set in at an early stage, hastened probably by an acute attack of gonorrhœa, from which the patient was suffering on admission. This was completely arrested for a time by frequent irrigations with different disinfectants. Two months after admission the patient stated he could feel the flow of water from the irrigator into the bladder, and this regained some power, the urine being occasionally expelled with force, though unconsciously to the man. After some months slight reflex action of the leg-muscles could be obtained by the galvanic battery, or by tickling the soles of the feet, and tended to increase to the last. Latterly he began to raise both elbows to the level of his head, and flex the left arm sufficiently to enable him to touch his face with his fingers, but he could not bring the arms back to his side. Rotation of the head became quite free and painless, and considerable pressure could be borne over the seat of fracture without causing discomfort. The temperature rose irregularly till the evening of Aug. 16th, when it reached 103·2°; after that date it gradually became normal or subnormal, and, excepting occasional short and sudden rises, remained so until the last week. The action of the bowels from first to last remained involuntary. About the middle of April, 1887, his appetite, which had been surprisingly good, began to fail, the urine became fetid, vomiting set in, the bedsores began rapidly to reappear, and on April 28th urine ceased to enter the bladder. Vomiting then became incessant, and on May 2nd, 1887, the patient died."

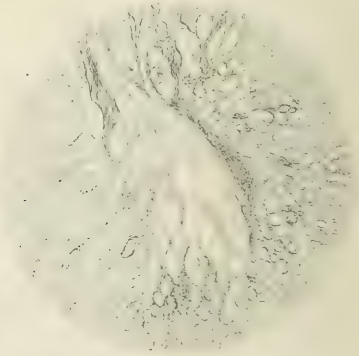
"At the *post-mortem* examination the cervical vertebræ were removed. The kidneys were completely disorganised, the right one being almost entirely converted into one fetid abscess. The ureters were much dilated and thickened, and deeply stained. The bladder was also much hypertrophied and stained, and contained only a little fetid pus. There was nothing special to remark about the thoracic organs. The brain was not examined."

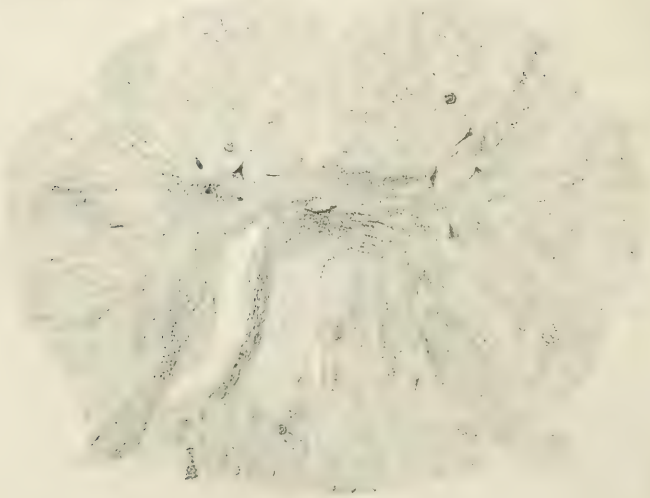
Only the cervical portion of the cord was available for examination. Opposite the projection of the body of the fifth vertebra it was much flattened and softened, its membranes partially adherent, and still blood-stained.

Sections of the cord made at the level of the second cervical









DESCRIPTION OF PLATE III.

To illustrate Mr. Silcock's case of Syringomyelia. (Page 18.)

From drawings by R. Ostlere, M.B.

FIG. 1.—Section of cord at the level of the second cervical vertebra (drawn under 1″ obj. but reduced). It shows general sclerosis, a slightly dilated central canal, and the elongated cavity in the right postero-external column.

FIGS. 2 and 3 (drawn under 1″ obj.) show the progressive stages of excavation and cavity formation in the newly-formed connective-tissue growth of the posterior column at the level of the lower portion of the sixth cervical vertebra, the fully-developed cavity being depicted in figs. 2 and 4.

FIG. 4.—Section of cord at level of sixth cervical vertebra, showing cavity in its right posterior column and general sclerosis. Drawn under 1″ obj. reduced.

vertebra show, even to the naked eye, an elongated slit-shaped cavity, lying immediately inside and parallel to the right posterior root (Plate III, fig. 1). Examined under a low power the cord in this situation shows general sclerosis and disorganisation, most marked in the right posterior column, that is to say, in the neighbourhood of the cavity. The walls of the latter are formedof finely reticulate connective tissue with abundant nucleation ; its lumen is irregular in shape, and presents no trace of a lining membrane, indeed, its inner surface is very ragged or frayed, when seen under a high power. There does not appear to be any connection between this cavity and the central canal, which is somewhat dilated but in other respects normal. The cavity reaches forward into the central grey matter of the posterior commissure, but does not encroach upon the adjacent posterior root. Fibrous claws or processes extend from the sclerotic tissue surrounding the cavity into the posterior root, the central grey matter, and neighbouring portion of the posterior column, gradually shading into and merging with the increased neuroglia found in this region of the cord. The cervical cord above the level of the second vertebra was unfortunately not reserved for examination.

At the level of the sixth vertebra the cavity is still seen, though considerably smaller in size, and somewhat differently disposed. Thus, its anterior end is placed posteriorly to the grey matter in the anterior part of the column of Burdach, whilst its posterior end lies in the posterior median column, so that its long axis lies nearly parallel with the posterior median fissure. The structure of its walls in this situation is essentially the same as obtains at the level of the second vertebra. Still lower, that is at the lowest portion of the cord submitted to me, the cavity ends in a mass of felted or reticulate sclerotic tissue, which is found at the site corresponding to that of the cavity, namely, in the right posterior column. But here very evident signs of excavation, as if by a process of mucoid softening, are apparent (Plate III, figs. 2 and 3), and ending in the formation of a well-marked cavity, (Plate III, fig. 3). From this mass fibrous processes are sent out into, and blend with, the surrounding neuroglia, which everywhere is in great excess.

The presence of a cavity of the nature and in the situation of that found in the present instance, has been ascribed to the persistence of a portion of that cavity which, in the embryo, extends as a fissure between the posterior columns to the periphery of the

cord, and becomes enclosed at a later period by the transverse development of the posterior columns, a view which would seem to be true in certain cases; and no doubt the fact that these cavities almost unexceptionally have their seat in the posterior columns is highly significant. But against this theory in the present and similar instances, are the arguments that the slit-like cavity has no appreciable connection with the central canal, that it exhibits no traces of a lining of cylindrical epithelium cells, and that the various stages in the development of the cavity out of sclerotic tissue by a process of softening and excavation is clearly demonstrable from an inspection of the specimens exhibited. Again, it is difficult to imagine the existence of such a cavity in the cervical cord with the absence of any manifestation of aberrant nervous phenomena (an assumption which is necessary in the present case) if the lesion be considered of the nature of a congenital defect; yet, as Westphal observes, "syringomyelia can no longer be regarded as quite independent of changes in the central canal, but a certain relation to such changes must be acknowledged."

January 3rd, 1888.

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9. *A case of Charcot's disease of the knee-joint.*

By W. Collier, M.D., and G. N. Pitt, M.D.

The left knee-joint of a female, aged 41, who died in the Littlemore Asylum of general paralysis, December 15th, 1887. The patient first came under observation as an in-patient of the Radcliffe Infirmary, Oxford, in August, 1885, at which time she had well-marked symptoms of locomotor ataxy, which had first appeared about twelve months previously. There was a strong history of syphilis, but none of either gout or rheumatism. No change in the joint observed. After a short stay she left the hospital, but was readmitted sixteen months later, in January, 1887, when she stated that in the interval she had been quite unable to walk about, and had been confined to the house. The left leg was

now observed to be swollen and œdematous, and the knee-joint distended with fluid, but no other change beyond the effusion could be discovered. The ataxy was so extreme that the patient was quite unable to stand without being supported on either side. She was discharged at the end of the month. Six months later, in August, 1887, she was admitted to the Littlemore Asylum, when Mr. Pilkington found that, in addition to effusion into the joint, the bones were freely movable in every direction, movement causing no pain, but being accompanied by marked grating. She died December 15th of the same year. On an examination of the joint it was found that enormous loss of bone had occurred, the external condyle of the femur had entirely disappeared, and the crucial ligaments had been destroyed, yet a large amount of new bone had been formed in the immediate neighbourhood of the destruction. All these changes had occurred in about six months, between the time of her leaving the Radcliffe Infirmary in January, 1887, and her admission to the Littlemore Asylum the following August. This specimen would go far to disprove the opinion held by Mr. Jonathan Hutchinson and others, that the changes are due to the anæsthetic condition of the joints, which allows patients to use and move a rheumatically inflamed joint in a way in which no other inflamed joints are used, for all these changes occurred during a time in which the patient was practically bedridden, and seldom moved the limb. One other point is worth noting, that in this patient the joint affection commenced at a very much later period than in the cases observed by Charcot, for although the symptoms of ataxia had existed for at least two and a half years, the joint affection only came on during the last year of her life.

January 17*th*, 1888.

Description of the spinal cord by Dr. Pitt.—The portion of spinal cord which was submitted for microscopical examination measured nine inches, and consisted of the lower part of the cord. The membranes over the posterior surface are slightly thickened.

The cord is small and distorted owing to extensive shrinkage of the tissue which has taken place. In the upper dorsal region there is a slight depression on both sides of the cord, but more marked on the right side along the surface of the cord in the region of the anterior nerve-roots. There is a depression along the line of exit of the posterior nerve-roots, which becomes very marked

in the lower dorsal region on the right side. In the lumbar region the shape of the cord is normal.

In the *mid- and upper lumbar regions* the posterior columns in their posterior two thirds are most severely sclerosed, and but few nerve-tubes persist ; in the posterior third, none. There are fibres, however, in a small biconvex area in the middle line, and scattered fibres along the line between the median and posterior columns. The anterior third of the outer columns are but slightly affected, and there is a scattered sclerosis of the anterior third of the median columns.

The severe sclerosis at the periphery extends slightly beyond the posterior roots, and in the lateral and antero-lateral tracts there seems to be a scattered diffuse slight degeneration near the surface, but none higher up the cord.

The anterior nerve-roots are healthy, the posterior are sclerosed. The cells of Clarke's column appear normal.

In the upper lumbar region the anterior horns differ considerably in shape, the left being round, and the right excavated anteriorly and not so broad.

In the *lower dorsal region* the area of sclerosis is similar to that in the posterior columns of the lumbar region. There is marked deformity and shrinkage on the right side; Clarke's column is almost destroyed, only two cells remaining (compared with twelve on the opposite side) ; the posterior median septum is deflected to the left, so that the distance of the posterior roots from the septum is one fourth greater on the right than on the left.

In the *mid-dorsal region* the sclerosis is extreme in the external root-zone, in the posterior third of the whole column, and not quite so severe on either side of the middle line as far forwards almost as the posterior commissure. There is a diffused sclerosis over the rest of the column. Clarke's column on the right side is much affected. In the upper dorsal region the distribution of the sclerosis is similar. The section is much distorted; there is a deep depression corresponding to the exit of the right posterior root. The cells of Clarke's column on this side are diminished in number, and the projecting lateral process of grey matter is absent on the left side.

Throughout the dorsal, and to a less extent in the upper lumbar region, the right anterior horn is narrower and not quite so large as the left; the motor cells are rather less numerous, and are smaller than those in the left.

The changes may be summed up as—

1. Extreme sclerosis in the posterior root-zone, *i. e.* in the posterior part of the external posterior column.

2. Sclerosis of Goll's column extending further forwards in the lumbar than in the dorsal region, together with slighter diffuse changes in the rest of the posterior columns.

3. Partial atrophy of Clarke's column on the right side.

4. The motor cells of the right anterior horn are smaller and fewer than those on the left side, and the horn itself is smaller, but these changes are only slight.

5. Distortion of the surface of the cord, the main feature being a wedge-shaped depression between the posterior and lateral columns on the right side.

6. Some of the posterior roots show degeneration.

The brain showed chronic inflammatory changes in the meninges and cortex. *May* 15*th*, 1888.

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10. *Lipomatous tumour* (*? sarcoma*) *of the spinal cord.*

By F. Charlewood Turner, M.D.

This specimen is the spinal cord of an obese female aged 54, who had had gradually-increasing paresis of the lower limbs for twelve years before her death, the weakness dating from six weeks after her last confinement (a still-birth). She was admitted into the London Hospital with a fracture of the left femur, caused by a fall in getting out of bed. She then had also a deep bedsore over the sacrum and pyrexia, which continued more or less until she died, five weeks afterwards, from pneumonia and œdema of the lungs. She had much fatty infiltration and atrophy of the heart, and fatty liver. In one kidney was a branched calculus. The fracture was ununited.

There is a lipomatous tumour about the middle of the dorsal region of the cord, which is of the size of a large filbert nut, and forms a well-defined fusiform expansion of the substance of the cord at that part. The cord above appears normal; below the

tumour it has softened and has almost all disappeared as far as the lumbar region, the greater part of which is still entire.

The tumour is free from attachment to the external membranes of the cord, and the outer layer of the pia mater sheathing the cord stripped off easily from a portion of it removed for microscopical examination.

In the transverse section of the tumour, in the preparation, it is seen that the left half of it consists wholly of adipose tissue, the cord appearing to have been pushed to the right and somewhat indented by the pressure of the growth. But inspection of the thin section of the tumour handed round with the specimen, shows that the cord has been almost wholly destroyed by infiltration with a similar growth, an open network only being left with its meshes filled with fatty tissue. This network is seen on microscopic examination to consist of interwoven strands of fibrous tissue, rather profusely studded with oval and elongated nuclei and leucocytes, especially in the more central part of the tumour, and enclosing remains of nervous structures. Slender tracts of the same nucleated fibrous tissue are seen traversing the adipose tissue, in which also small clusters of nuclei appear between the fat-cells.

At the part of the section of the cord nearest to the surface of the tumour (*i. e.* to the right of the cord), the remains of the white columns of the cord are distinctly recognisable, though granular, possibly from defective preservation of the specimen. Towards the centre of the section, where the fibro-nuclear growth is more abundant, there are spaces between the interlacing fibrous bands which have a more translucent appearance, and in which a reticulum is discernible, resembling that of the cord, but in which no nerve-fibres are distinguishable.

There is no trace of the central canal or of the central grey matter of the cord discernible.

There is much thickening of the sheath of the cord, the outer layers being fibrous, the inner layers fibrous and nucleated like the connective-tissue growth infiltrating the cord, with which they are continuous. And the line between the purely lipomatous mass and the cord is marked out by interrupted tracts of nucleated connective-tissue growth, which are evidently parts of the inmost layer of the sheath of the cord. Embedded in the extra-medullary part of the growth is a bundle of medullated nerve-fibres, evidently a nerve-root emerged from the substance of the cord. This part of the

tumour is also traversed by tracts of nucleated tissue, which are mostly seen to be connected with vessels.

It seems to me that there are two possible views that may be taken as to the probable pathology of this tumour of the spinal cord, which would appear to be of a very unusual character.

It might be supposed that in the first instance a lipoma originated, in a very obese subject, in the pia mater ensheathing the cord between the more superficial and the deepest layers; that this growing exceedingly slowly, began to cause injurious *compression* of the cord twelve years before the patient's death, and that an infiltrating fibrous connective-tissue growth so produced became the seat of fatty infiltration, owing to the same abnormality of nutrition which caused the general obesity of the body.

But it is, I think, more probable that this tumour, though having the appearance of a simple lipomatous tumour, and consisting almost entirely of adipose tissue, is essentially of a sarcomatous nature, a lipomatous sarcoma or fibro-sarcoma, of very slow growth, originating in the pia mater, and compressing and invading the substance of the cord.

If of this nature the growth is allied to other observed instances of tumour of the cord, in which lipomata would appear to be unknown,[1] though they occur in relation to the dura mater.

November 1*st*, 1887.

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11. *Alcoholic paralysis of the phrenic, pneumogastric, and other nerves.*

By Seymour J. Sharkey, M.D.

[With Plates IV and V.]

E. R., aged 32, married, was admitted into St. Thomas's Hospital under my care on August 29th, and died on September 25th, 1887. Her family history was unimportant. She had always

[1] In vol. xxvii of the 'Transactions' of the Society (p. 19) Dr. Gowers has recorded a case in which a lipoma was found attached to the surface of lower extremity of the cord, and embracing it on one side; but the growth had not invaded the substance of the cord.

been a delicate woman, and seven years previously had been treated as an in-patient at St. Thomas's for what the husband called "the liver complaint and yellow fever." For some years she had been in the habit of drinking hard, and had taken principally whisky and beer. She had suffered from diarrhœa for three years, and about five months before her admission she had an attack of hæmoptysis. Ever since then she had been losing flesh and getting weaker. For three months she had been evidently losing power in her legs, and had appeared "queer in her head." For two months she had vomited nearly everything she took, and had lived almost entirely upon beer. She had also complained of numbness and cramp in her legs.

On admission, she was found to be fairly well nourished; her complexion was sallow, and venous stigmata were marked upon the cheeks. She understood well enough what one said to her, but she was confused and incoherent in her answers. Pulse was 130 and weak; the heart's dulness normal, and the sounds free from murmur.

The respiratory sounds were harsh, but there was no evidence of pulmonary disease.

The tongue was thickly coated with yellow fur, the liver somewhat enlarged and hard. Urine contained no albumen.

The legs were wasted, the muscles along the front of the tibia having suffered most, but all the muscular movements were very deficient in power. She could neither walk nor stand. There was marked superficial and deep tenderness. The knee-jerks were absent, but the plantar reflex was present. Temperature was normal. For the first week but little change occurred; the pulse remained rapid, she had delusions at night, cramps in the arms and legs, tremors of the tongue and lips, and extreme hyperæsthesia of the legs, with moderate flexion at the knee-joints. The temperature was but little above the normal.

On September 9th, at 2 p.m., she had an attack of shivering in which her face became livid, the temperature rose to 102·8°, great dyspnœa occurred, and the respirations increased to 58, and the pulse to 180, in the minute.

She was given fifteen minims of tincture of digitalis and half a drachm of aromatic spirits of ammonia every two hours.

Next morning (September 10th), the temperature had fallen to 99·6° F., the respirations were 38, and the pulse 140. She took

fluid food well. As her nights were restless she was given thirty grains of bromide of potash and fifteen grains of chloral, which had the desired effect.

On September 13th, she had two attacks of intense dyspnœa, and had to be propped up in bed. The temperature rose to 102° F., and the pulse to 150 per minute. The chest was examined during the attack and a few bronchitic râles were heard, but it was then noticed for the first time that the diaphragm was completely paralysed.

When I made my visit on September 14th she had another attack, and I made the following note:—"Has attacks of paroxysmal dyspnœa often accompanied with pyrexia and shivering, and with very rapid pulse and respiration. Frequently too she suffers from difficulty of swallowing, but no fluid comes back through the nose. Respiration is at present at the rate of 40 a minute, and pulse 140. The diaphragm is quite paralysed, and the abdomen is drawn in during inspiration, while the upper part of the chest acts with unusual energy."

On the morning of the 15th she began to spit some blood, and on examination impairment of resonance was found at the left apex, and some crepitation.

When going round on the 16th, I ventured to suggest that the extreme rapidity of heart's action, increased in paroxysms, together with the difficulty in swallowing, which seemed to be due to some pharyngeal defect, depended upon neuritis of the pneumogastric nerve.

The hæmoptysis soon passed off, but the paralysis of diaphragm and rapidity of heart's action continued to the end of her life. Ether, ammonia, and digitalis were given every six hours, and she seemed to improve somewhat. A good idea of the condition of respiration and heart's action is given by the following note, which my clinical clerk, Mr. Tuppen, wrote on September 22nd:—"Digitalis has now been given in fifteen-minim doses every six hours for one week, and in comparing her condition during this week with that during the week before digitalis was given, we find that during the week ending on September 15th the pulse-rate on the twelve occasions on which it was taken was as follows: 180, 150, 140, 144, 132, 150, 126, 132, 128, 150, and 138, the highest being 180, the lowest 126, and the average 143. In the week ending September 22nd, the rate on thirteen occasions was as

follows: 100, 132, 120, 140, 144, 140, 150, 140, 126, 130, 132, 130, 132; the highest being 150, the lowest 100, and the average 132.

"In the first week respirations averaged 41, in the second 32. During the first week the patient had attacks of severe dyspnœa, at least on three occasions, but during the second she has had none."

On September 23rd, clear evidence of disease at the apex of both lungs was obtained.

On September 26th, I was in the ward when the Sister hurriedly called me to the bedside of the patient. She had a sudden paroxysm of intense dyspnœa, with great rapidity of pulse, and before anything could be done her face became blanched, and on listening to the chest I found her heart had stopped beating, although she still continued to breathe; in a few seconds she was dead.

Post-mortem examination twenty hours after death.—Body well formed and well nourished. Diaphragm on level with fourth ribs.

Heart somewhat pale. Slight hæmorrhage beneath the endocardium of left ventricle, no fatty degeneration detectable, valves normal, right side filled with firm clot.

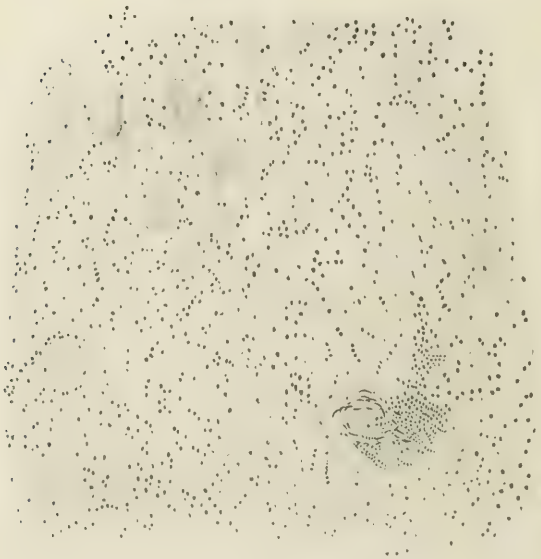
Lungs: Both lungs adherent to the chest wall at apices, and both the seat of rather extensive tubercle. In the right there were small cavities at the apex, and scattered tubercles even in the lower lobes.

Liver large, rather firm, apparently in early stage of cirrhosis.

Kidneys large and very hyperæmic, but otherwise healthy.

Nervous system: Spinal and cerebral membranes healthy. Brain normal throughout. In the dorsal and lower cervical region of the cord there was softening, which seemed to be pathological and not *post mortem*. The upper cervical region, cervical enlargement, lumbar enlargement, pons and medulla were healthy, as far as the naked eye could judge. There was a slight superficial hæmorrhage in the dorsal region. None of the muscles or peripheral nerves presented any appearance of disease, apart from diminution in size of some of the former.

After careful hardening, a microscopical examination was made of the following parts, with the results about to be detailed, viz. the parietal convolutions of the brain, the medulla oblongata, the cord on a level with the origin of the fourth and fifth cervical nerves, the cervical enlargement, the dorsal region, and the lumbar enlargement, the pneumogastric nerve and heart, the phrenic



DESCRIPTION OF PLATE IV.

To illustrate Dr. Sharkey's case of Alcoholic Paralysis. (Page 27.)

From drawings by M. H. Lapidge.

FIG. 1.—Lumbar enlargement of spinal cord, showing the vascular changes of inflammation, and degeneration of nerve-cells.

FIG. 2.—Section of popliteal nerve showing intense inflammation.

nerve and diaphragm, the popliteal nerve, the tibialis anticus and peronei muscles, the liver and kidneys.

Motor convolutions: In this portion of the nervous system slight inflammatory changes were observable, there being an excess of leucocytes; the large nerve-cells were ill defined in outline and their nuclei stained very badly. But although the changes present were sufficient to be termed pathological they were slight in degree.

Medulla oblongata: Here the same clear but slight inflammatory phenomena were observable, both vascular and productive of degeneration of large nerve-cells.

Spinal cord (Plate IV, fig. 1): In the spinal cord the changes were much more advanced, and were most marked in the lumbar enlargement and on a level with the fourth and fifth cervical nerves. In the former situation there were numbers of leucocytes in the grey matter, and some of the vessels were full of them. The nerve-cells in the anterior cornua were swollen, shapeless opaque, with but few processes and with very ill-stained nuclei. The conditon of these large nerve-cells reminded me of that of voluntary muscular fibres in the earlier stages of degeneration. On a level with the origin of the fourth and fifth cervical nerves the condition of things was very similar though somewhat less marked.

In the dorsal region, and on a level with the seventh cervical nerve signs of inflammation were present, but when compared with those in other regions of the cord they were trivial.

Phrenic nerve (Plate V, fig. 1) and diaphragm: The phrenic nerve at its entrance into diaphragm was the seat of intense inflammatory changes. Leucocytes were very numerous, and accumulated even in masses worthy of the name of miliary abscesses; they were seen in large numbers both in and around the vessels. A similar condition was also observed in the trunk of the nerve higher up. The muscular substance of the diaphragm was evidently inflamed and altered in physical properties. The connective-tissue spaces between the fibres were beset with leucocytes, and considerable aggregations of them were seen both in and around the vessels. Some of the muscular fibres were pretty well striated, other swollen, opaque, and destitute of striation.

Pneumogastric nerve and heart: All along the trunk of the nerve were numbers of leucocytes, and many vessels were filled with them and surrounded by those which had escaped from their channels.

The heart showed very evidently the vascular changes of inflammation; but the striation of its muscular fibres was fairly well preserved.

Popliteal nerve (Plate IV, fig. 2): This nerve presented marked inflammatory changes. Many vessels both in the sheath and in the nerve-bundles were filled and surrounded by leucocytes. Axis-cylinders were frequently seen swollen, and the white substance of Schwann displaced by a substitute which distended the neurilemma and took the logwood stain.

Tibialis anticus and peronei muscles (Plate V, figs. 2 and 3): Intense vascular phenomena of inflammation seen, and muscular fibres altered, so that generally longitudinal striation alone was visible, and often the fibres were wholly destitute of any striation.

Liver was in a condition of early but well-marked cirrhosis and fatty infiltration, and there was a very unusual degree of dilatation of the small vessels in its interior. a condition which reappeared in the kidney to a remarkable extent. Nearly all the small vessels between the convoluted tubes and elsewhere in the kidneys were dilated, and could be seen under the microscope forming a well-marked plexus. There was some cloudy swelling of the epithelium lining the tubes, but no other evidences of disease.

The case which I have just narrated is one of alcoholic paralysis in a comparatively early stage, and one that was, probably up to the time of death, still progressive. All the microscopical evidence obtained from an examination of the nervous and muscular systems points to the presence of an acute inflammation. The nerve-trunks of the lower limbs and the muscles controlled by them were affected, so was the phrenic nerve and its muscle, the diaphragm. The fact that the pneumogastric nerve and the heart's muscle were both the seats of inflammation shows that the vagus probably bears the same relation to the nutrition of the myocardium that the motor nerves of the limbs bear to the muscles which they supply. Other cases of alcoholic paralysis affecting the diaphragm have occurred at St. Thomas's Hospital, of which Dr. Bristowe has recorded one. They recovered, however, and the exact nature of the lesion could not be definitely proved. In Dr. Bristowe's case too it was pretty clear from clinical symptoms that the recurrent laryngeal nerves were affected, and the superior laryngeal as well. In my case the whole trunk of the pneumogastric was involved, and the symptoms which made it possible to diagnose this lesion are interesting, viz.

DESCRIPTION OF PLATE V.

To illustrate Dr. Sharkey's case of Alcoholic Paralysis. (Page 27.)

From drawings by M. H. Lapidge.

FIG. 1.—Longitudinal section of phrenic nerve showing intense inflammation.

FIGS. 2 and 3.—Section of peroneus muscle showing great number of leucocytes, and disappearance of transverse striation from muscular fibres.

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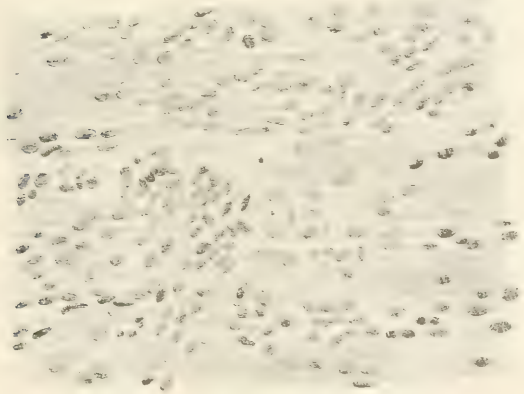

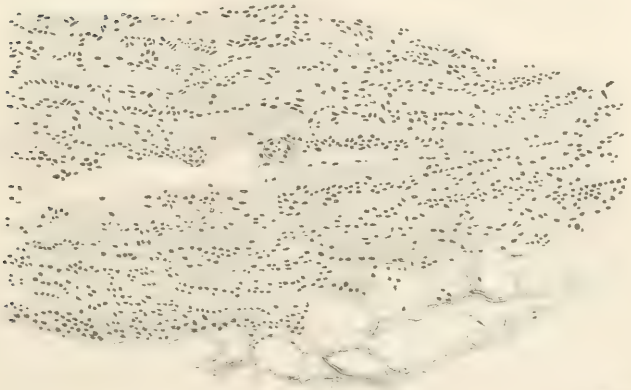

the extreme and constant rapidity of heart's action increased in paroxysms, without evident heart disease, and the difficulty in getting food past the pharynx. Another point, which is not recorded in the notes, but which I remember well, was great feebleness of voice. This, however, seemed to be possibly due to the want of respiratory power owing to the paralysis of diaphragm.

The microscopical examination of the nervous system in this case showed that the inflammatory processes were most intense in the peripheral nerves and their muscles, less intense in the spinal cord, but nevertheless well marked, and that the brain and medulla oblongata had suffered least. Recent investigations have rather tended to rivet the attention on the peripheral nerves alone in alcoholic paralysis; but it must not be forgotten that alcohol may affect the central nervous system as well, and that severely.

The extreme congestion of the kidney and liver might be a phenomenon due to peripheral paralysis of their vaso-motor nerves, or of the centres of the latter, which are well known to be located in the medulla oblongata. Everyone who has made many *post-mortem* examinations must have been struck by the frequency with which diseases due to alcohol are found side by side with phthisis. And this connection seems to be almost the rule in cases where the poison attacks peripheral nerves. Five cases of this disease have been recorded by Dr. Hadden in the 'Transactions' of this Society, and in four phthisis was certainly present; in the fifth case there was "softening broncho-pneumonia," so that in all there was serious lung disease. Dr. Hadden says, "I am inclined to think that it is not a mere coincidence." The case which I have brought before the Society shows the extent to which the vagus can be affected by alcohol; but it is not at all improbable that it may be affected often enough in a much slighter degree. There are two conditions at least required for the production of phthisis, viz. firstly, some impairment of the nutrition of the lung, and secondly, the tubercle bacillus. To me it seems by no means unlikely that the first of these conditions is produced in alcoholic cases by trophic changes lighted up in the lung by peripheral neuritis, either of the branches of the pneumogastric or of other nerves which supply these organs. At any rate, when we consider the present state of our knowledge as regards the effects which alcohol may have on the body, we must allow that its tendency to affect peripheral nerves opens out for it an almost unlimited field of action. *April 17th*, 1888.

II. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

Diphtheria (two cases of). (*Card specimen.*)

By R. G. Hebb, M.D.

Case 1.—*Post-mortem examination.*—Sanio-purulent discharge from nose; thick tough leathery membrane from posterior nares downwards to cords, from which it spreads onwards to the ultimate ramifications of the bronchi, but becoming thinner and thinner, it finally ends as a suppurative bronchitis, which in the right lung has given rise to a "diphtheritic" pneumonia of the middle lobe. Microscopical examination shows the ordinary histological appearances of the "pseudo-membrane" in which near the free edge are a few isolated colonies of bacilli.

Case 2.—*Post-mortem examination.*—Glands about jaw much enlarged. Mucosa of fauces and upper pharynx much swollen, of a purple colour, and the seat of irregular patches of greyish-yellow membrane, which in places is very thick. The mucosa of the larynx and bronchi is in a similar condition, but the membrane is less thick. The tissue subjacent to the membrane is "raw and red." Microscopical examination shows the ordinary histological appearances in the "pseudo-membrane," the central zone of which is occupied by a network of small bacilli. This network seems under a $\frac{1}{12}$th apochromatic to be quite continuous. At intervals there are ovoid expansions in the course of the network.

The significance of the microbes in these two cases is to be reckoned from their number and distribution, for in the one there are but a few colonies situate near the free margin, in the other there is a continuous network of micro-organisms extending from the free surface as far as the central zone, where they are most evident. In the former case the membrane is thick and leathery and continuous; in the latter it is sloughy and discontinuous. It

is possible that the first case might have been what was known formerly as "croup," and the latter as "putrid sore-throat." In both cases there is distinct resemblance between the micro-organisms, but a great difference between their distributions.

December 6th, 1887.

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2. *A Case of pemphigus involving the larynx.* (*Card specimen.*)

By John R. Lunn.

Charles F—, aged 39, had always been a healthy man until March 20th, when, during the evening, while at home after his day's work, he complained of great irritation of his skin, and on undressing found that his skin was entirely covered with blisters.

State on admission.—His body was covered with bullæ, varying in size from a pin's head to a large walnut; some of the bullæ were in a state of suppuration. Those on his head were smaller than on the extremities. His temperature varied from 99° to 103·6° F., and he seemed very ill. He was put on large doses of arsenic, with a full diet, and his health improved for a few days. On April 10th, his throat became very sore, and his mouth was covered with bullæ, making it difficult for him to swallow, and as he had pain in his stomach with diarrhœa his medicine was stopped. On April 13th, he had a severe attack of dyspnœa, and coughed up a large piece of diphtheritic-looking membrane, after which he was relieved for a time, but his breathing and general condition gradually became worse, and he died on April 16th.

At the *post-mortem* his larynx was found studded with patches of white membrane, which extended to the bifurcation of the trachea. The mouth and tongue were covered with ulcerated patches. No other changes were found in the body. No ulcerated patches were found in the intestines. *May 1st*, 1888.

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3. *Three cases of malignant disease of the air-passages.*

By Felix Semon, M.D., and Samuel G. Shattock.

The clinical portion of these cases is by Dr. Semon, the pathological by Mr. Shattock.

Subglottic alveolar sarcoma in a patient eighty-one years of age.—On September 15th, 1887, I was requested by Dr. Warren, of Selhurst, to see an old gentleman, aged 81, on account of considerable dyspnœa.

He had enjoyed perfect health until the beginning of the previous winter, when he had fallen ill with hoarseness and gradual loss of voice. No acute illness preceded these symptoms. There was no pain and no dysphagia, but abundant expectoration of frothy, sometimes sanguinolent sputum. There were no phenomena indicative of disease on the part of the central nervous system, the thoracic organs, or œsophagus. Within the last few days there had been much loss of strength, and in the night before I was consulted, a sudden and considerable aggravation of the dyspnœa. Iodide of potassium had been used for some length of time without benefit.

When I arrived, the patient could only with the greatest difficulty be induced to receive me, and could not be persuaded to submit to a laryngoscopic examination. Paralysis of the glottis-openers had been diagnosed a fortnight previously.

Present state.—A much emaciated man; respiration stridulous but not sonorous in character. Inspiration somewhat more difficult than expiration, but the latter also much impaired. Voice quite aphonic. The larynx makes moderate respiratory excursion; is not broadened; slight tenderness on pressure, but a little more on the right than on the left side.

On the right side of the cricoid there is a hard swelling apparently intimately connected with the cartilage, and of the size of a hazel-nut, which has grown considerably, if not indeed appeared, during the last few weeks; it is slightly tender on pressure. No enlarged lymphatic glands are anywhere to be felt.

The patient drank some water without difficulty in my presence. Heart-sounds normal. Thorax slightly emphysematous. No signs of aneurysm. Pulse rather weak, but regular.

After excluding other possibilities, I stated that in my opinion there was most probably malignant disease of the larynx, the nature of the dyspnœa and the aphonia rendering bilateral abductor paralysis highly improbable. I recommended tracheotomy, but the patient could not be induced to undergo the operation.

Two days afterwards death took place from asphyxia. Permission was given to examine the larynx. There were large clusters of glands on both sides of the larynx, especially on the right, and more posteriorly in the furrow between the larynx and œsophagus. These could not be felt during life; they did not compress the larynx, the trachea, or the œsophagus.

The morbid growth forms an extensive though nowhere very prominent swelling, extending over the greater part of the laryngeal cavity below the cords, which are involved in the disease and lie almost in apposition. Inferiorly the growth ceases at the lower margin of the cricoid cartilage, but the posterior wall of the cavity is free. The extreme thickness of the intra-laryngeal portion of the growth is one centimetre.

The tumour extends through the cricothyroid space, and appears externally in front of the larynx. On the right side the extra-laryngeal portion overlaps the ala as high as the apex of the thyroid notch, and infiltrates the pyramidal lobe of the thyroid gland; in its most prominent part it is slightly less in thickness than the intra-laryngeal portion.

The mucous membrane over a large area of the growth is villous. This condition is quite independent of any ulceration of the growth, and it is one that might possibly in certain cases be of much clinical importance.

A similar villous condition is at times observable in the mucous membrane of the bladder in connection with sarcomatous tumours (see a case by one of us [Mr. Shattock] in the Pathological Society's 'Transactions,' 1887).

Histology.—A vertical longitudinal section of the intra-laryngeal growth, taken to a depth of 5 cm., displays a coarse alveolar structure, the trabeculæ of which are in places abundantly infiltrated with small round-cells. The alveoli are filled with multiform cells, the nuclei of which are considerably larger than the nuclei of the cells infiltrating the trabeculæ.

The tumour must be classed as an alveolar sarcoma, from the fact that delicate fibres pass from the trabeculæ amongst the cells,

and that in some places a fine reticulum is visible between the epithelioid elements.

Remarks.—The case is noteworthy in two respects. In the first place, sarcoma of the larynx is a comparatively rare disease, the total number of cases hitherto reported not exceeding fifty.

Secondly, the patient is, with one exception, the oldest in whom malignant disease of the larynx has been observed.

In the case referred to, the patient was eighty-two years of age; it is recorded by Dr. Semon in the 'St. Thomas's Hospital Reports' for 1883, vol. xii. A case of malignant disease in a patient of eighty is recorded by Dr. Hopmann, of Cologne.

It is to be observed that in both these instances the disease was carcinoma. So far as sarcoma is concerned, no case occurring at so advanced an age as the present has yet been published.

Mr. Butlin, in his concise little treatise on 'Malignant Disease of the Larynx,' cites in the list of cases of sarcoma one observed in a patient of seventy-four years.

Case 2. *Epithelioma of right half of larynx, with epitheliomatous insula on the left vocal cord.*—On October 12th, 1887, I was consulted by Mr. R. W—, aged 57, on account of hoarseness which had commenced about six months previously without any known cause, had increased gradually, and at the time of consultation had led to nearly complete aphonia. Within the last few weeks some shortness of breath on exertion had been added. There was no pain, no dysphagia, no cachexia, no emaciation.

The larynx was somewhat broadened, but there was no tenderness on pressure, and no enlargement of the glands in the neck.

On laryngoscopic examination, it was seen that the whole of the right half of the larynx except the epiglottis was converted into an immovable, red, infiltrating mass, from which only a small portion of the thickened and immovable vocal cord projected. The left vocal cord did not pass the mid-line on phonation, and therefore did not reach the right cord, which was fixed immovably in the cadaveric position. In its anterior portion there appeared also some slight congestion and thickening. The other organs were quite healthy. No history or other marks of syphilis.

Ten grains of iodide of potassium ordered three times a day. A week later, as no improvement had taken place, the dose was increased to fifteen grains.

On the 21st of October, I had a consultation with Mr. Butlin, who shared my views that the disease was malignant, and who felt a distinct thickening on the right half of the cricoid cartilage.

On the 27th, the disease was observed to have made distinct progress anteriorly. No ulceration was perceived in the larynx, but the vocal cords did not separate entirely in front during inspiration. It was impossible to say with certainty whether this was due to increase of the swelling on the right side, or whether the disease had extended to the left side of the larynx.

After the failure of iodide of potassium the patient had been given to understand the nature of the disease, and his chances with regard to palliative and radical treatment had been clearly explained to him[1]. The rather rapid increase of the disease within so short a time rendered it very doubtful in our minds whether radical operation could still be recommended at the end of the month, since we were not sure as to the extent of the disease. Nevertheless the patient made up his mind to submit to partial or, if necessary, even total extirpation of the larynx, and the former was performed by Sir William Mac Cormac, who has kindly sanctioned the publication of the case, on November 1st, 1887, with the assistance of Mr. De Meric, Mr. Middleton, and myself, Mr. Tyrrell giving chloroform. Tracheotomy was first performed, the thyroid isthmus being split between two ligatures. The trachea was opened, and Hahn's compressed sponge-cannula inserted. After ten minutes (allowed to give time for the sponge to swell) the thyroid cartilage, which was ossified, was divided in the middle line with a small saw, and the halves of the larynx turned apart. Dr. Semon's electric lamp was now introduced into the larynx, when, in addition to the main mass on the right side, a small hard mass was observed in the anterior commissure. The posterior wall of the larynx was seen to be free.

The right half of the larynx was removed with knife and scissors; there was but slight hæmorrhage. When the lamp was now again introduced, it was seen that on the lower surface of the left vocal cord in front, but not continuous with the mass in the anterior commissure, there was an isolated infiltration of the size of a split pea, not adherent to the cartilage; this was excised by a circular incision in the healthy parts around it.

[1] With regard to this point, see a paper by Dr. Semon in the 'Clin. Soc. Trans.,' 1887, vol. xx, on "A Case of Partial Extirpation of the Larynx," &c.

The wound was plugged with iodoform gauze, and the edges of the wound united, a drain being left in.

The patient passed a good night. On the following morning (temp. 97°, pulse 84) the dressing was changed and nutrient enemata given, the patient being also fed through an œsophageal bougie with a mixture of milk, brandy, and eggs.

At noon there was a sudden attack of suffocation, and Sir William Mac Cormac substituted for the sponge-tube a common tracheal cannula covered with india-rubber.

At 1.15 I saw the patient. The temperature, which had risen without any rigor in the course of the morning to 100°, had gone down to 97°, but the pulse was 140 and very weak; considerable perspiration; respiration rapid; tracheal stertor. No dulness in front of chest; breathing vesicular. In the course of the afternoon the frequency of respiration increased to 48; pulse 150, temp. 100°. Face livid; dulness posteriorly at both bases, and bronchial breathing; pleuritic friction at right base.

At 9.30 there was a fresh attack of suffocation, the night being subsequently passed rather quietly. At 4 o'clock on the morning of the 3rd another attack of suffocation occurred.

At 9 a m. face more livid, temp. 102°, pulse 160, filiform. Respiration very hurried; jactitations; patient comatose. He rallies from time to time from the coma; is free from pain, but always thirsty. Death occurred about noon. No *post-mortem* was obtained.

The parts removed comprise all those appertaining to the right side of the larynx, excluding the epiglottis.

There is a widespread swelling beneath the mucous membrane, most marked in the region of the true cord, in which latter situation the growth is exposed by ulceration.

Externally there is a somewhat similar swelling beneath the cricothyroid muscle, which is stretched over its surface and infiltrated by it; the growth extends upwards anteriorly for about a centimetre over the outer surface of the ala of the thyroid, and that it has passed beyond the middle line appears from the divided surface of the tumour in this situation. The intra- and the extralaryngeal growths are continuous.

From the left vocal cord there was removed at the same operation an isolated lenticular tumour, ·7 cm. in diameter; the tissues around this growth were lax and apparently quite healthy.

Histology.—Horizontal sections of the isolated tumour involving the left vocal cord show a growth ·2 cm. in thickness, ·7 cm. in horizontal diameter. The tumour has the structure of a typical carcinoma.

The branching cell-columns have no lumen, and they can be traced to the free surface, towards which the cells are of distinct squamous type; in the deeper parts the cells are of smaller size; there is no difference in form between the peripheral and the more central elements of the columns, and no "nests" occur. A portion of the surface of the tumour is in a state of necrosis, and the superficial epithelium is for the most part wanting.

There are evidences of inflammation along the whole of the part of the cord removed, in small-cell infiltration close beneath the surface, and catarrhal changes in the columnar transitional epithelium.

At one of the extreme ends (the commissural) of the specimen, the morbid growth again appears; there is an interval of ·5 cm. between this portion and the cancerous insula first described. The investing epithelium between them is of the transitional columnar kind; in the region corresponding with the growth last mentioned it is stratified squamous. The mucous glands are not involved.

The growth is therefore an "epithelioma" in the sense that it has arisen in connection with investing epithelium, as distinguished from glandular carcinoma.

The larger tumour of the opposite (right) side of the larynx shows an exactly similar structure.

Remarks.—This case illustrates the necessity of arriving at a definite diagnosis in certain cases of malignant disease of the larynx from clinical symptoms only, without the aid of the microscope, for the infiltrating nature of the growth in this case would have rendered the intralaryngeal removal of a fragment for the purposes of microscopic examination impossible.

In spite of its fatal issue (apparently from septic pneumonia) the case deserves recording, since, as stated by one of us (Dr. Semon) in a previous communication, it is only by publishing every case of extirpation of the larynx, whether successful or not, that a more complete consensus of opinion can be arrived at in regard to the value of this operation for carcinoma of the larynx.

Lastly, as to the isolated growth on the left vocal cord. Is this due to contact or to metastasis? Infection by contact is a question

which is still *sub judice*, and it is difficult by clinical observation only (as in so many other cases) to arrive at any indisputable conclusion ; the direct proof must be delegated to experiment. There is no theoretical reason, however, why contact carcinoma should not occur, were the surface opposed to the primary growth excoriated or ulcerated, as might arise from its contact with decomposing cancerous discharge ; for as the secondary glandular growths in carcinoma arise from the direct transference of elements from the primary tumour, there seems no reason why the direct implantation of infective epithelium to a granulating surface might not lead to the growth of a second tumour as well, seeing also that the grafting of normal epidermis is so readily effected on a similar surface.

That carcinoma might be communicated to an intact epithelial surface is also conceivable, though in either case it might be held in argument that the spot opposed to the primary tumour was prepared only by the ensuing inflammation to become cancerous, for the same general reason which lay at the bottom of the original disease.

Prof. von Bergmann[1] has recently shown, at the Berlin Medical Society, a case in which there was carcinomatous disease on exactly opposite spots in the upper and lower lips. Such an association, as Sir James Paget remarks, is exceedingly rare. One reason of its infrequency may perhaps lie in the fact that the pain arising from carcinoma induces the patient to keep the lips apart, both when awake and during sleep.

We may add that, so far as we know, the only fact in the history of *laryngeal* carcinoma similar to that under discussion has been recorded by Dr. David Newman, of Glasgow.[2]

Case 3. *Intratracheal carcinoma continuous with carcinoma of the thyroid.*—A man, aged 39, was admitted on July 8th, 1887, into George Ward, St. Thomas's Hospital, under the care of Dr. Ord, to whom, as well as to Dr. Sharkey. who in Dr. Ord's absence took charge of the ward, we are obliged for permission to use the case.

His family, as well as his previous history are unimportant.

He has had blood-spitting and cough for the last two years, both of which have been very constant and severe. Ten months ago he had very severe attacks of hæmoptysis, bringing up, according to

[1] 'Berliner klin. Wochenschrift,' No. 47, p. 891, 1887.

[2] "Discussion on Cancer." Reprint from 'Glasgow Medical Journal,' 1886, p. 95.

his statements, pints of blood at a time. He had been for two years in the Birmingham Infirmary; has lately lost much flesh. Two months ago, the blood-spitting began to be worse. There has never been much night sweating. Voice lost within a week previous to admission; felt feverish during the last few days. He has had great difficulty of breathing for about the last ten months.

Present state.—On admission, an emaciated-looking man, complaining of troublesome cough, hæmoptysis, and loss of voice. Voice reduced to a whisper, occasionally breaking out into a high-pitched note. Inspiration markedly prolonged and stridulous. Expiration fairly quiet. Paroxysms of dyspnœa occasionally, when the stridor is exaggerated.

Lungs: Deficient resonance at apices; sinking-in of chest above the clavicle. Deficient movement during respiration. At right apex in front tubular breathing and whispering pectoriloquy. Voice resonance exaggerated; a few crepitant râles. At left apex, in front, a few crackling sounds. Behind, slight tubular breathing on both sides with increase of vocal resonance and whispered voice sounds. Bases of lungs normal.

Heart: Dulness nearly obliterated. Apex-beat not localized; sounds feeble but normal.

Liver: Dulness begins at sixth rib; edge not felt.

Abdomen normal. Urine 1026, clear, acid, no albumen. Pulse 96.

Tongue slightly furred, dry, red.

July 10th.—Paroxysmal cough and stridor, and difficulty of breathing at times, but not so severe as on admission; has been spitting up bright blood as well as muco-pus. Sweats at night. Pulse 80. Fixity of trachea during breathing. Just above the sternal notch is a round, hard swelling, more marked on the right side, apparently surrounding the trachea; pressure on this lump causes cough. The patient was first ordered an expectorant, and later iodide of potassium.

10th.—The patient was seen by me, when the following note was made regarding the laryngeal condition:—"Right cord fixed near middle line; left moves well. Large ulceration on anterior wall of the trachea corresponding to the projection outside about the third to fifth ring of the trachea. Diagnosis: Most probably tubercular perichondritis of tracheal cartilages."

14th.—Urine alkaline, contains much triple phosphate.

20th.—No crepitations to be heard. Dulness at apex not quite complete.

21st.—Patient complains of pain when he talks. Larynx moves fairly in swallowing.

24th.—I saw the patient for the second and last time. "Tracheal ulceration looks cleaner than on the 8th, otherwise no change."

25th.—Food taken well and without pain, but it always returns. Pain on coughing, just above the sternum. No hæmoptysis for two days.

August 10th.—Dr. Sharkey made the following note:—"On auscultation the air is found to enter poorly into both lungs, but hardly at all into the left. There is no definite evidence of lung disease; but the tracheal sound obscures everything and makes diagnosis of the pulmonary condition impossible. Two small hard lumps were felt on right side of trachea near the sternum, and another rising up from thorax as patient swallows. There is also thickening about the trachea here. Left vocal cord moves freely; the right is slightly adducted but not abducted. No laryngeal disease."

During the next few days the condition was varying; slight improvement.

22nd.—The sputum was examined but no bacilli found. Patient much troubled with cough; has spat up considerably more blood than for the last fortnight. Pain in front of chest and at back of the left side. Breathing has been bad. Continued loss of weight, and, for the first time, rise of temperature, which had been normal or subnormal up to this time.

During the next few days considerable hæmoptysis, pains in chest, irregular rise in temperature, pulse varying from 108 to 124. The hæmoptysis constantly increased, and on September 4th the patient died, the temperature having been rather irregular during the last few days, respiration hurried, pulse 136. Neither ergot nor tannic acid checked the bleeding.

Post-mortem examination.—Into the trachea, about 1·5 cm. below the lower border of the cricoid, in the mid-line and on the right side, there projects a smooth ovoidal growth, 2 cm. in its longer vertical diameter. On carrying a section through this tumour and tracheal wall into the circumjacent part of the thyroid gland, the morbid growth is seen to be continuous with a widespread extension of similar substance beneath the tracheal mucous membrane. This submucous infiltration extends for 3 cm. in the vertical direction, and attains a maximum thickness of ·3 cm. within the line of the

tracheal cartilages, and it leads to some diminution of calibre in the neighbourhood of the projecting intratracheal process of the growth. The tumour-substance spreads between the tracheal cartilages (which are completely embedded in it) into the thyroid gland. The gland is, as a result, inseparably incorporated with the trachea for the distance of 3 cm. or more, as above mentioned. The upper parts of the lateral lobes of the thyroid are unaffected; the lower parts are so infiltrated that their limit is unrecognisable, the growth passing beyond over the sides and front of the trachea.

The right recurrent laryngeal nerve was found at the *post-mortem* examination to enter the mass of malignant disease which lay round the trachea. The lower lobes of both lungs were affected with acute pneumonia. The other organs were healthy.

Histology.—Vertical sections of the projecting intratracheal growth show a uniform structure of closely-set, narrow, winding, tubular spaces, lined in some places with cubical in others with subcolumnar and columnar epithelium. This difference in epithelial form is unconnected with any difference in the size of the spaces. The epithelium is everywhere formed of a single layer of cells, and there is everywhere a well-marked lumen; where it is typically columnar no ciliated border is discernible under $\frac{1}{12}$ homogeneous immersion. A few of the spaces contain an unstained, finely reticular substance, in which there are minute orange-red granules and a few large pigment carrier-cells; others of the spaces are distended by hæmorrhage, and this has clearly led also to the contents first noticed. The intertubular stroma is small in amount and of ordinary connective tissue.

The tracheal epithelium is intact over the growth and is altogether distinct from the epithelium of the latter, even where the columnar-celled spaces of the tumour in the subjacent corium approximate quite closely to that of the trachea.

In sections carried through the submucous extension of the growth and into the extratracheal portion a similar structure to that described obtains, the amount of fibrous tissue being, however, much more considerable in these situations. The growth, preserving these characters, is traceable between the tracheal rings, and its thyroidal origin becomes decisive within a short distance of the outer side of the trachea, where the spaces become larger and more spherical, and where the typical, homogeneous, pale yellow, colloidal substance fills some and extravasated blood others.

That the case is not one of invasion of normal thyroid tissue by the ingrowth of another kind of tumour appears from the fact that a perfect continuity exists between the intratracheal growth and that without the trachea where the thyroidal character becomes more pronounced; and this suffices to determine what is in other respects a debateable question, viz. whether the tumour has originated in the trachea and subsequently invaded the thyroid, or whether it has originated in the thyroid and secondarily invaded the trachea? That the growth, in any case, has not arisen from the investing epithelium of the trachea is clear from the fact that the epithelium of the tumour is disposed throughout in a single layer and that the spaces have everywhere a distinct lumen.

It may still be questioned, however, whether the growth may not be a columnar-celled carcinoma that has arisen in the mucous glands of the trachea; and but for the distinct thyroidal characters of certain parts of the growth it would not be easy to disprove such a view.

The new growth, therefore, is to be considered as a carcinoma of the thyroid which by its extension has involved the trachea and come to project within it.

Remarks.—One of the reasons why this case has been put on record has already been mentioned in the concluding portion of the histological description, viz. the difficulty of deciding whether the growth was of tracheal or of thyroidal origin. The clinical history presented considerable difficulty. The presence of malignant disease of the thyroid was not suspected during life, and the idea that the tracheal affection was of malignant nature presented itself to Dr. Sharkey only during the last month of the patient's life. Previously to this the idea of tubercular mischief of the lungs and trachea appeared the more probable; there was the long history of hæmoptysis, emaciation, night sweats, and cough; there were the pulmonary signs described; there was paralysis of the right vocal cord, which was explicable on the hypothesis that the recurrent nerve had been involved in pleuritic thickening at the apex of the right lung, an event by no means rare in tubercular disease of the right lung. There was, moreover, the tracheal ulceration which, added to the external thickening of the trachea, naturally suggested tubercular perichondritis of the tracheal cartilages. Had the intratracheal projection been situated somewhat higher up it would have been possible to see that one had to do not with an ulceration

merely but with an ulcerated tumefaction. The disease of the thyroid remained latent during life.

Pathologically, the case is interesting from the fact that the disease appeared in the trachea in the form of so well-defined and considerable a tumour; this is a rare occurrence. When malignant disease commencing in the œsophagus or thyroid gland invades the trachea there is usually an ulcerating infiltration only. Cylinder-celled carcinoma, lastly, is a rare form of thyroid tumour. Wölfler,[1] who gives a full description of its formation (loc. cit., p. 787, *et seq.*), mentions only seven cases described by various authors.

April 3rd, 1888.

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4. *Lung from case of fatal hæmoptysis in an infant.*

By F. D. Drewitt, M.D., for H. P. Cholmeley, M.D.

The child, aged 1 year and 8 months, was admitted into the Victoria Hospital for Children on March 13th, 1888. There was dulness and bronchial breathing in left axilla, and a history of cough and wasting since measles three months before.

On March 20th she was seized with sudden, profuse, and immediately fatal hæmoptysis.

Post-mortem.—Organs were blanched. Trachea filled with frothy blood. Posterior mediastinal and bronchial glands much enlarged (forming solid mass round trachea). Some of these glands were caseous. Gland at bifurcation of trachea was much enlarged.

On opening the left bronchus from behind it was found to terminate in an irregular hard-walled cavity filled with caseous material and blood-clot. It was about the size of a walnut and occupied the upper part of the lower lobe, and lower part of the upper lobe.

On washing away the contents, a vessel the size of a small crow-quill was seen crossing the cavity; on the vessel there was a small perforation the long diameter of which was in the axis of the vessel; blood was flowing from it. On injecting water into the pulmonary

[1] 'Archiv für Klin. Chir.,' vol. xxix, Heft 1 and 2, 1883, "Ueber die Entwickelung und den Bau des Kropfes."

artery a stream flowed at once from the rupture. No other cause for the hæmoptysis could be found.

The left bronchus was rough and ulcerated, and at one point on its anterior wall there was a communication with a caseous gland.

Small grey tubercles were scattered throughout both lungs, and there were some in kidneys and spleen. The lung and trachea were shown. *April 3rd*, 1888.

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5. *Two cases of mediastinal cancer.*

By H. Handford, M.D.

[With Plate I, fig. 2.]

Case 1. *Carcinoma of the mediastinum and left lung, causing perforation of the œsophagus, and death from hæmorrhage from the left auricle, which was involved in the growth.*—The patient, J. E—, a stockinger, aged 45, was an old soldier and had suffered from some liver affection in India. There was no appearance or definite history of syphilis. His health began to fail, and he suffered from cough about six months before his admission under my care into the Nottingham General Hospital on January 26th, 1888, when he complained of loss of flesh, pain between the shoulders and at the lower end of the sternum, difficulty of swallowing anything but fluids, and of cough with slight purulent sputum frequently tinged with blood. The difficulty of swallowing was the most urgent symptom. "For the past fortnight he has been unable to get any solid food down; when it gets part of the way down it sticks and he has tried to wash it down with fluid, but has always had to vomit it up again." He swallowed water readily in my presence, and neither then nor at other times did swallowing give rise to paroxysms of coughing. An œsophageal bougie was passed without any difficulty on two occasions at one sitting, and was arrested on the first occasion twelve and a half, and on the second twelve and three quarter inches from the dental arch. He thought that his swallowing was better for a few days afterwards, and succeeded in taking minced meat. He gradually, however, lost ground, and by

February 9th could not swallow even water. On February 12th, he brought up about a pint of blood. It came up slowly in small quantities at a time, and was of a bright arterial colour. The hæmorrhage ceased for about ten hours, but began again, and he spat up another 10—15 oz. during the night of the 12th—13th, and died at 10 a m. on the 13th.

At the inspection on February 13th at 3 p.m., five hours *post mortem*, rigor mortis was well marked, especially in the arms, but the body was still warm. On opening the thorax, the left lung was found to be very firmly and universally adherent to the costal parietes. The right lung was very voluminous but not adherent. The heart was small, and the pericardial sac contained a small quantity of fluid blood, and a small elongated recent black clot. There were a few long narrow adhesions between the epicardium and the pericardium, apparently of some weeks' duration at least. The pericardium on the left side was so firmly adherent to the indurated left lung that the heart could be lifted out of the pericardial sac, like a nut out of its shell, without the walls collapsing. The source of the hæmorrhage was the appendix of the left auricle, which was adherent to the pericardium, and communicated by a small irregular aperture both with the pericardial sac and with a sloughing cavity at the root of the lung in the mediastinum. All the cavities and the main vessels leading to and from the heart contained partially decolourised blood-clot. There was no valvular disease. The heart was removed *in situ* together with the larynx, trachea, œsophagus, and both lungs. The left lung was removed with much difficulty, and only by the free use of the knife.

Between the base of the lower lobe and the diaphragm was a smooth space containing recent lymph, evidently a localised diaphragmatic pleurisy. The œsophagus was nine inches long from the lower border of the cricoid cartilage, and when opened disclosed no stricture, as was anticipated, but at five inches from the upper extremity two small ulcerated openings, roughly oval in shape, with their long axes parallel with the œsophagus. At five and three quarter inches from the cricoid there was a similar but much larger opening one and a quarter inches long. The margins of the opening were thin, black, and sloughing. The openings communicated with a large cavity with gangrenous walls, into which opened the trachea and the right and left bronchi; a great part of the walls of these latter were destroyed and disintegrating. The cavity

was bounded by the œsophagus and trachea, the roots of the lungs and the posterior aspect of the heart. Besides the free communication with the œsophagus and trachea already described, the appendix of the left auricle was also involved as part of the walls of the cavity, and communicated with it by a small irregular opening.

The walls of the œsophagus were not in any place involved in the new growth, which occupied the whole of the lower lobe, and one third of the upper lobe of the left lung. The pleura was specially thickened, and prolongations spread from it into the lung. On section the affected part of the lung was of cartilaginous hardness, airless and grey in colour, resembling in appearance a chronic pneumonia, and showing a sharp, limiting, white border. The rest of the upper lobe was engorged and friable but crepitant. In the right lung there was some hypostatic pneumonia, but with the exception of the root it was not involved in the growth. No rupture of any of the pulmonary vessels could be discovered, or any other source for the hæmorrhage except the left auricle. It is also to be remembered that the blood was bright in colour as would be the case from the left auricle, and not dark as it must be from the pulmonary artery. The glands at the root of the neck were enlarged.

The stomach was very small and contracted, and contained disintegrated blood.

The liver weighed 48 oz. and contained numerous white secondary deposits. The liver-tissue was very friable, and some of the nodules could be quite readily shelled out, though they were not distinctly encapsuled. The average size of the nodules was about three quarters of an inch in diameter, but they varied from the size of a pea up to two inches in diameter. In all there were about a dozen. There were no other secondary deposits. The kidneys appeared healthy. The brain was not examined.

On microscopic examination of the nodules in the liver a typical alveolar stroma of scirrhous carcinoma was found. In the sections of the lung the stroma was less distinct except where it was collected into bands which permeated the lung in all directions. Some of these were plainly visible with the naked eye, while others were much finer, and required a low magnifying power. The cells varied much in shape, but were mostly of an epithelial type. The growth in the pleura consisted chiefly of fibrous tissue.

A very similar case is recorded by Impaccianti in 'Lo Speri-

mentale' for January, 1888, tomo lxi.[1] In this case a mediastinal growth involved the left auricle, and caused pressure on the œsophagus leading to perforation. Difficulty of swallowing was the chief symptom, and the patient died of inanition after being totally unable to swallow for several days.

A third case, somewhat similar to the above two, is the following, of which I show drawings and microscopic sections.

Case 2. *Carcinoma of the lung; death from hæmoptysis; secondary growths in the muscles and in both kidneys.*—S. R—, a well-developed man, aged 40, was admitted under my care into the Nottingham General Hospital on July 4th, 1887, complaining of cough and spit, pain under the scapula and at the epigastrium after eating. He had suffered from cough more or less for twenty years, and five years ago brought up a large quantity of blood. Nearly two years ago he lost his voice for three months. He has been unable to work for eighteen months, during which time he has lost 2 st. in weight, and has suffered much from muscular pains. He had a very abundant frothy and muco-purulent sputum, occasionally streaked with blood. His temperature was hectic, varying from 98·6° to 101° F., and occasionally much higher (104·4°). There was impaired percussion resonance over the greater part of the right lung, but especially over the lower lobe posteriorly. He had a very irritable cough which the most varied treatment failed to relieve.

The diagnosis was felt to be obscure, but it was thought that the physical signs pointed to bronchiectasis, except that he did not, as is usual in bronchiectasis, periodically bring up mouthfuls of fœtid sputum.

He gradually lost ground, and on July 28th complained of severe pain in the right side, which proved to be the beginning of a pleuritic effusion for which he was tapped on August 18th, and 30 oz. of slightly turbid fluid withdrawn, giving him much temporary relief.

I was away from home during August, but on examining the patient on September 3rd I found a swelling the size of an egg in the upper third of the right humerus, apparently periosteal in origin and very painful; another nodule about the size of a cherry in the right deltoid; others, of about the same size, in the subcutaneous tissue on the inner side of the left upper arm and on

[1] An abstract of this case appears in the 'London Medical Recorder,' 1888.

the inner side of the left thigh. There were smaller nodules in the episternal notch and on the scalp.

He went home unrelieved on the 7th, and on the 17th had a copious hæmoptysis which proved speedily fatal.

At the inspection, about thirty-six hours *post mortem*, the growths in the muscles, &c., were found but little increased in size, and some of them were removed for microscopic examination. The growth on the right humerus was found to involve the bone very deeply, and the humerus was fractured during the examination, though no unusual force was used. The right pleural cavity contained about a pint of clear straw-coloured fluid; the pleura was very much thickened and almost as hard as cartilage. The lung-tissue was very much disorganised and contained numerous cavities, one of which was filled with bloody fluid and numerous recent clots. It was probably the source of the fatal hæmorrhage. At the root there were several growths which spread along the bronchi and blood-vessels into the lung. One of the large pulmonary veins was found completely embedded in new growth. On the left side there was no fluid in the pleural cavity; the lung was voluminous, not adherent, and did not contain any new growths or tubercle.

The pericardium was not involved; the heart was small and all the valves healthy.

The liver and spleen were normal in appearance and contained no growths.

The kidneys were large and friable; the capsules stripped easily, except where they were involved in the numerous new growths, varying in size from a pea to a large marble, and about ten or twelve in number, with which each kidney was studded.

The mesenteric and retro-peritoneal glands were not enlarged.

On microscopic examination, the appearances of the growth at the root of the lung and of the secondary growths in the muscles bore a very close resemblance. The cells were irregular in size and shape and of epithelioid character, and there was an abundant stroma. In places the alveolar structure was well marked. In fact, the microscopic examination pointed to carcinoma. The clinical picture, especially the absence of marked enlargement of the lymphatic glands (except the bronchial), the involvement of the bone (humerus), and the nodules in the muscles and subcutaneous tissue, is rather more suggestive of sarcoma.

The growth in the root of the lung appeared to be the original one, but whether it commenced in the bronchial glands, bronchial mucous membrane, or in the connective tissue, there was no evidence to show. Considering the abundance of the secondary growths, the small mass of the primary growth was surprising. The pleura was much involved, and the growth at the root of the lung sent prolongations along the blood-vessels and along the interlobular septa in a manner highly characteristic of scirrhous carcinoma, especially as that form of tumour is seen in the mammary gland.

All the other growths, except that in the humerus, viz. those in the kidneys, muscles, and subcutaneous tissue, were of moderate size and distinctly nodular. I think there can be no doubt that they were secondary. The growth in the humerus was larger and of older date, as indicated by the extensive destruction of the bone shown by its fracture, but it did not attract attention on his admission two and a half months before his death. I think it must have been of later origin than the growth of the lung, though this is open to question.

The character of the growth was very similar in the two cases that I have brought forward, and the description of the lung in Dr. Impaccianti's case would have applied exactly to the first of mine. The numerous fibrous processes radiating into the lung from the pleura at the root, and the dull grey pneumonic appearance of the lung, were the common features.

The majority of mediastinal growths that have been described recently have been called sarcomata. I think there can be no doubt that the two cases I have brought forward are true carcinoma.

The tissue in which a tumour grows appears to influence very considerably the arrangement of its constituent elements, and especially is this true of the lung. Hence, sections from the growths in the lung did not in either case present nearly such a typical arrangement of cells and stroma as did those from the secondary growths, which, I think, we may safely take as a true index of the nature of the primary tumour. *April 3rd*, 1888.

6. *Malignant disease of bronchial glands.*

By G. N. Pitt, M.D.

1. *Sarcoma of bronchial gland invading bronchus.*—Wm. G—, aged 70, admitted January 31st, 1888. Was found lying unconscious, and was brought up to the hospital. On admission, he was conscious and was able to sit up. He was put to bed, but insisted on sitting on the edge and groped about under the bed; he did not see a porringer when given him. He gradually became comatose and died eight and a half hours after his accident, the limbs being rigid and the right side paralysed.

Post-mortem.—Cerebral hæmorrhage on the left side of the brain bursting on to the surface; submeningeal hæmorrhage; extensive bruising of the brain. Four fractures radiating from a point on the left temporal bone, and crossing the posterior branch of the middle meningeal artery, but none of the fractures extended to the base of the skull.

There was bronchitis. A soft vascular small-celled sarcomatous growth involved the bronchial glands between the bronchi, forming a mass one and a half inches long by one inch in diameter, and lying below and in front of the left bronchus, which it had invaded slightly, so as to form a small swelling on the inner surface, the mucous covering not being ulcerated; the lumen was but slightly diminished. Adjacent to the growth were four glands containing calcareous débris. No other growth was found in the body.

Heart 14½ oz; left ventricle much hypertrophied; right ventricle slightly thickened. Mitral valve incompetent; old vegetations on the aortic valves. Indurated viscera.

Kidneys 11 oz., not granular, but the arteries were thickened.

2. *Carcinoma of bronchial gland, with secondary growths in opposite lung.*—Mary N—, aged 67, admitted September 7th, 1887, under Dr. Pye-Smith. A delicate woman who for a month has had dyspnœa, especially when she is lying down. The lymphatic glands in the posterior triangle of the neck are enlarged and painful. The right side of the chest moves less than the left, and but little air enters on that side; the voice-sounds are diminished in front. The other organs are normal.

September 9th to 20th.—Better entry of air into right chest.

October 3rd.—Decidedly deficient entry of air again on right side. A hard mass, tender on pressure, can be felt one and a quarter inches above and to left of umbilicus, which transmits aortic pulsation. The patient has all along taken but little food. At the end of December this lump, which was dull on percussion, increased up to three and a half inches in diameter, and the diagnosis of carcinoma ventriculi was made. The patient suffered severely with epigastric pain, vomited, emaciated, and was extremely ill. She was delirious at night.

January 12.—Temp. 100°—101.° F. Pleuritic rub on left side. She lingered on, gradually becoming more feeble till her death on February 27th.

Post-mortem examination by Dr. Shaw.—52 oz. of fluid in the two pleural sacs, more in the right than in the left. Left pleura thickened.

The right bronchus was ruptured in removing it just at its commencement; around it was growing a small tumour obstructing the tube so that it would just admit a probe. The mucous membrane was not ulcerated, nor had the growth invaded the bronchus. The bronchi beyond were slightly dilated.

The left lung was healthy but for some pus in the tubes and a few scattered nodules of growth. There was no diffusion of the growth along the mediastinal glands, but the right supra-clavicular glands were involved.

Heart 11½ oz.; normal. Aorta atheromatous.

All the abdominal organs were normal, and nothing could be found to account for the supposed abdominal tumour except a rather unusual prominence of the vertebral column. Liver 37 oz. Spleen 5 oz. Kidneys 8 oz. Capsule adherent, surface granular.

Microscopically, the bronchial growth was found to be a carcinoma consisting of large cells with large, deeply-stained nuclei embedded in a fibrous stroma forming a hard mass, so that it should be considered a glandular scirrhus. The growth surrounded and severely compressed the right bronchus, which it almost occluded, but it was not definitely invaded. The bronchial glands, and possibly also some of the adjacent lung-tissue, were the seat of the growth. It appeared that the growth had originated in the mass by the bronchus, and that the left lung and the cervical glands were involved secondarily. As far as it was possible to judge, the growth

originated in the lymphatic gland, but one could not be absolutely certain that this was the case, as the lung might have been the starting-point. Although primary carcinomata of the lymphatic glands are described, it is *a priori* more probable that carcinoma has originated in epiblastic structures. Still, such a case as the present is some evidence in favour of a primary seat in the glands, as there were physical signs of compression of the bronchus for nearly six months before her death, and the nodules in the opposite lung were very small. Without, however, insisting upon this, I record the case as an example of primary growth commencing below the bifurcation of the trachea.

The secondary involvement of bronchial glands by malignant growths originating in other parts of the body are frequently met with, but I am desirous of drawing attention to cases such as those I am bringing before you this evening, where the primary growth is in the bronchial glands or immediately adjacent thereto.

Scarcely any allusion in text-books is made to primary growths in the glands, and but few cases are recorded, yet they cannot be so very rare, as I have myself met with three cases; and I have at the present time a man under observation in whom I can find evidence of nothing beyond signs of pressure on one bronchus, with a bleeding surface in the respiratory tract, probably at the same spot. I have now watched this case for over a year.

These growths, starting in the bronchial glands, seem to progress but slowly, in one of my cases being found accidentally, and the other patient, after being five months in the hospital, died with double hydrothorax. *May* 15*th*, 1888.

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7. *Primary cancer of pleura.*

By G. N. Pitt, M.D.

Sarah P—, aged 61, admitted December 3rd, 1887, under Dr. Goodhart. One year ago she caught cold, she has since had cough, shortness of breath, and pain in the left side; the pain has persisted, and the cough is troublesome.

On admission, the patient was very thin; she has clubbed fingers;

the right hand is livid, cold and clammy; pulsation in vessels equal on the two sides. The circumference of the right chest is fourteen and a quarter inches, of the left thirteen inches. There is deficient movement of the left side, with tenderness in the axilla, and increased pain when the arm is raised; dulness all over, extending to margin of sternum; breath-sounds scarcely audible, only a few râles and rhonchi after coughing, with distant cracklings in the axilla. Tactile vocal fremitus much diminished; œgophony behind. On the right side the chest is resonant, T. V. F. normal, breathing supplementary, with a few râles at the base. Heart's impulse in fourth space, one inch inside nipple, sounds normal. Pulse 88. Other viscera normal.

December 7th.—A syringeful of blood-stained serum withdrawn by a puncture in eighth left space.

11th.—On waking, patient had a difficulty in articulating and swallowing. Left arm is powerless.

12th.—Right pupil larger than left; left facial paralysis. Left arm very weak. No impairment of power in leg. Impaired sensation in left hand.

22nd.—Patient is dull and slow of apprehension. The left hand is quite powerless now. She passes everything under her. An attempt to aspirate the left chest failed.

January 8th.—Severe dysphagia.

11th.—She has wasted greatly since admission. To-day she is lying on her back, groaning incessantly; she pays no attention to anything; dysphagia. Paralysis of right arm and hand to-day; she can still move her legs. She died at midnight.

Post-mortem.—Body very emaciated.

Brain 44 oz. A decolourised clot in right middle cerebral artery, without any definite softening. The vessel was blocked beyond the origin of the branches to the capsules and the third frontal convolution. There was 26 oz. of blood-stained serum without any lymph or clot in the left pleura.

The left lung was airless, pigmented, and splenized from compression, being firmly adherent by growth to the wall of the chest both behind and above; elsewhere it was quite free. The lung did not extend below the fifth rib behind; the visceral pleura over the front of the lung looked healthy, it was not thickened, rough, or opaque.

The costal pleura was dense, opaque, and white, one fifteenth of

an inch thick, with numerous small, oval, and circular depressions due to contraction (similar to what is seen with a dense capsulitis of the liver). The whole of the left pleura lining the ribs was uniformly invaded by growth, and formed a dense sheath. The pleura on the posterior surface of the lung was a dense fibroid mass, one third to one fourth of an inch thick, which nowhere showed any signs of invading the lung-trabeculæ, but which extended up to the clavicle, capping over the upper part of the lung.

There were secondary growths in the glands in the supra-clavicular region, along the œsophagus, between the bifurcation of the trachea, beneath the arch of the aorta, and along the pericardium. There were dense fibroid masses of growth, one third to one half of an inch across, in the diaphragm. All the growths were firm and non-succulent.

The growth partially constricted the aorta at the junction of the transverse and descending parts, and extended round the bronchi near the trachea and round one branch of the pulmonary artery on the left side, but did not invade the root of the lung.

There were numerous small nodules of growth, one eighth to one third of an inch, on the pericardium, chiefly on the anterior and upper part, and almost confined to the parietal layer. There were ʒij of blood-stained serum in the sac. There were small nodules beneath the visceral pericardium over the anterior wall of the right ventricle and along the right posterior auricular-ventricular fissure. The right side of the heart was otherwise normal. The aortic valves were not thickened, but from the corpus aurantii of each valve projected small warty vegetations. From two spots on the left mitral flap warty vegetations projected on the auricular surface; to one of these was attached a small recent clot. Weight of heart 7 oz.

On the jejunum there were five minute (one twentieth to one sixth of an inch across, and one-fiftieth of an inch thick), creamy-white, encapsulated secondary growths in the submucous tissue. They were scarcely palpable, were flat, and the largest showed a villous surface.

Liver 32 oz., small. There were two minute secondary growths. There were secondary growths in the glands along the aorta and iliac arteries extending to Poupart's ligament on the left side, and into the groin on the right. These were numerous but not large, and had been infected from the thorax by contiguity.

Spleen 2½ oz., healthy; kidneys 7 oz.; two small nodules in the right kidney.

There was well-marked lateral curvature of the spine concave to the left, the concavity being most marked about the first dorsal, and extending to the fifth, or beyond. There was a slight diffuse curve, beyond this, concave to the right, and at the last lumbar vertebra and sacrum a marked concavity to the left.

The outer condyle of the left knee was quite bare of cartilage, except one third of an inch at its inferior margin. The patella, except round the margin, was bare; the right knee-joint grated on movement. The great-toe joints were healthy.

The thoracic muscles were not infiltrated with growth. The veins and arteries of the right upper extremity were normal, and nothing abnormal was noticed in the brachial plexus, so far as it was examined, to account for the marked lividity and coldness of the hand during life.

Microscopically, the growth shows a fibrous stroma with many spindle- and round-cell nuclei containing elongated alveoli lined, some by cylindrical celled epithelium, others by glandular epithelium, and in some there are columns of cells with large nuclei. The thickened pleura consists of malignant growths with a thin layer of parallel bundles of fibrous tissue with elongated nuclei on the surface. In some sections there is a doubt whether the growth ought to be called a cylindrical-celled epithelioma or not, but the growths in the right lung are quite definite, and there are numbers of tubes lined by a very elongated cylindrical epithelium arranged characteristically.

The small submucous nodules in the duodenum when examined microscopically contain growth, but they are so minute and localised that they must be considered to be secondary; moreover, they are contained in well-defined fibrous capsules; nowhere do they infiltrate the adjacent tissues, or invade the mucous layer.

The costal pleura presented a very remarkable appearance, forming a thin uniform sheet of growth lining the chest wall; it was dense, and had by its shrinkage formed numerous small lacunæ such as are not uncommon with perihepatitis. The appearances presented in the *post-mortem* room of a uniform dense infiltration of the whole of the parietal pleura by growth pointed very strongly to its being the primary seat whence the disease had spread over

the apex of the lung. The masses of growth in the anterior mediastinum and in the abdomen appeared to be secondary growths in the glands, and a chain of infected glands could be traced from the groin up the aorta to the pleura. The visceral layer of the pleura was quite healthy over the left lung, except at the apex. The growth on the surface of the lung did not affect the root but rather the apex, and had not spread inwards; it did not appear to be the primary growth.

Every portion of the body was carefully searched for the primary source, and it appeared conclusive at the *post-mortem* that the pleura was the primary seat. When on microscopical examination the growth was found to be a cylindrical-celled epithelioma it was strongly suggestive of a primary growth elsewhere; the only possible sources were:

1. The submucous growths in the duodenum. These were very small flat multiple encapsulated plaques nowhere invading adjacent tissues, and were unquestionably secondary.

2. The mucous glands of the bronchi. The growth did not invade the root of the lung, while the growth at the apex of the left lung, and the small nodules in the right, were quite superficial, and did not affect any bronchi so far as one could judge.

While, therefore, the result of the naked-eye examination was opposed to these being the primary source, it could not be positively excluded.

The history of persistent left-sided pain for one year was most definite, and both this and the remarkable cuirass condition of one layer of the pleura might be explained by a primary cancerous degeneration, all the other growths in the body being such as are often found as secondary results.

As our knowledge on the subject is extremely scanty it seemed desirable that the case should be recorded as a contribution towards the subject.

Primary cancer of the pleura is very rare, but few undoubted cases have been recorded, and as to the microscopical character of such growths I have been able to learn hardly anything.

Lepine, in 'Bull. de la Société Anatomique,' 1869, reports a case, but it is by no means conclusive, of a child, aged 10, who was ill three months with cough and dyspnœa. The right chest was dull and there was deficient breathing. *Post mortem* there was found carcinoma of the pleura invading also the lung.

Dutil, in 'Gaz. méd. de Paris,' reports a case of primary cancer of pleura and pericardium.

Dieulafoy, in 'Soc. méd. des hôp.,' 1886, reports a case of primary cancer of the diaphragmatic pleura with growths in the left lung, liver, vertebræ, &c., in a man, aged 23, who for some years had violent pains in his chest, and was tapped for hæmorrhagic pleuritic effusion twenty-five times before his death. *May 15th*, 1888.

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8. *Pulmonary endarteritis.*

By George F. Crooke, M.D.

[With Plate VI.]

The specimens I have brought with me were taken from a patient admitted into the General Hospital in November, 1886, under the care of Sir W. Foster, by whose kind permission I am able to exhibit them to the Society to-night.

Clinical history.—The following is an epitome of the leading facts of the clinical history of the case, taken by Dr. Stacey Wilson, then resident medical officer:—The patient, a man aged 34, was a printing-ink maker by trade. His *family history* is unimportant. Regarding his previous history, he had scarlet fever when a child, but afterwards remained in pretty good health till five years ago, when an attack of rheumatism in his right shoulder confined him to the house for a week. His circumstances were fairly comfortable, and his occupation by no means laborious. There was no history of syphilis.

History of present illness.—His present illness, which dates about eighteen months previous to his admission, commenced with shortness of breath on exertion, and pains in his left side and back. During the first half of this period he continued at his work, but shortly afterwards he was obliged to give it up entirely and take to his bed for some days on account of his symptoms becoming gradually worse. From this time he was more or less constantly under medical treatment, both privately, and also as an out-patient

at two metropolitan hospitals, obtaining, however, only temporary relief. On October 12th, i. e. about three weeks before admission, he removed from London to Birmingham, and feeling himself worse, became at once an out-patient at the General Hospital, and was sent for admission on November 1st.

Condition on admission.—The patient was a well-built man, but short in stature. He complained of shortness of breath, pain and fulness in his abdomen, and swelling of face and legs. His temperature was 98°, pulse 84, regular but rather hard, and there was œdema of both legs below the knees.

Circulatory system.—The cardiac impulse was felt in the fifth interspace, three quarters of an inch external to nipple line, and a loud systolic bruit was heard over both mitral and tricuspid areas, transmitted to the axillæ; heard also, though not so distinctly, over the aorta, but not over the pulmonary area. Pulmonary second sound much accentuated.

Respiratory system.—Regarding the respiratory system there was no dulness, but the breath-sounds were high pitched and of a wheezing character, and there were a few moist râles heard at the bases.

Urine diminished in quantity, of a deep orange colour, contains albumen. Five days later the report on the urinary chart says: "Urine deep orange colour, turbid, with lithates, 1011 sp. gr.; quite a thick cloud of albumen; no blood or sugar; the microscope shows a few leucocytes, an occasional hyaline cast, and some spheroidal epithelial cells, probably renal."

Alimentary system.—Appetite often ravenous, complains of great thirst. Tongue dry, but not furred. Abdomen full and tympanitic; there is no ascites or enlargement of the liver. On the evening of the 10th he was not so well, became very restless, and said he felt as if he were being suffocated. Death occurred at 9 p.m.

Necropsy fourteen hours after death.—A well-developed and well-nourished subject, with livid swollen features and some œdema of the legs and feet.

Pericardium distended with 14 oz. of clear yellow serum.

Right heart much distended, with *post-mortem* coagula. The right ventricle much dilated and walls hypertrophied, measuring two-fifths of an inch in thickness; tricuspid orifice dilated, but valves normal. The conus arteriosus of the pulmonary artery as well as the artery itself, large and prominent; pulmonary orifice large; no

valve lesions; there was dark, loose *post-mortem* thrombus occupying both the main trunk as well as the two branches of the artery. The arterial wall was much thickened and hypertrophied and somewhat rigid, equalling in this respect the aorta, but there was no disease of the inner lining outside its distribution in the lungs.

The left auricle was much dilated, its walls hypertrophied, and its endocardial lining opaque and thickened. The mitral orifice was narrowed but the valve looked healthy, and there were no evidences either of former or recent endocarditis; the stenosis must therefore be regarded as congenital. Left ventricle slightly thickened, but its hypertrophy is quite insignificant relatively to that of the right ventricle.

Lungs bulky and not collapsed. On section the most striking appearances were presented by the medium-sized and smaller branches of the pulmonary artery, which stood out rigid and patent, like so many quills, and resembled at first sight thickened and dilated bronchial tubes. On following out some of the larger dilated branches near the root of the lung the internal coat was found to be the seat of nodose patches of endarteritis and atheroma. No distinct calcareous plates or spicules were met with, but the intima was roughened and uneven, and the seat of thickenings, that here and there were of a yellowish colour and undergoing degenerative changes. Of the branches of medium calibre some were thickened, others irregularly thinned and dilated, and the intima on both was affected with endarteritis. The smallest branches appeared as minute thrombosed points in the midst of little patches of greyish pigmented fibrous tissue.

Regarding the lung-texture itself it was intensely hyperæmic and dry-looking except at the bases, which were moderately œdematous. The systemic arteries—brachials, radials, and femorals—looked thickened, but there was no atheromatous change discoverable. Thoracic and abdominal aorta normal. Cerebral arteries at the base of the brain likewise appeared a little thicker than normal, but so far as the naked eye could detect, they were neither atheromatous nor fatty. There was much cerebral venous congestion.

Abdominal viscera: 23 oz. of serum in the peritoneal cavity. Liver 57 oz.; surface uneven, granular; capsule thickened. On section the appearances were those of advanced atrophic nutmeg liver. Spleen 6 oz., firm and congested. Kidneys 11 oz.; capsules slightly adherent; organs indurated and intensely cyanotic.

Microscopic examination of the lungs revealed the following conditions:

(1) Dilatation of the larger and medium-sized pulmonary arteries, much hypertrophy of the muscular coat, and bulgings of the intima caused by endarteritis and hyaline swelling, with fatty degeneration of the subendothelial connective-tissue cells. The ground substance of the swelling resembled hyaline fibroid material, *i. e.* consisted of a partly homogeneous and partly fibrillated structure, in which branched, spindle-shaped, and a few round-cells were embedded. Osmic acid preparations showed the former to be undergoing granulo-fatty degeneration (Plate VI, fig. 2).

(2) The smaller branches, viz. those distributed probably to individual pulmonary lobules, are affected with an obliterative endarteritis.[1] The periarterial fibrous tissue is sometimes extensively infiltrated with small round-cells, leucocytes, and lymphoid cells (fig. 3); sometimes it is seen to be much thickened and pigmented, and there is marked collateral hyperæmia of the venules around.

(3) The pulmonary veins are dilated, and there is great and general ectasis and engorgement of the pulmonary capillaries throughout the lung.

Remarks.—Pulmonary endarteritis is a rare affection, and its etiology is by no means a settled question. Thickening and dilatation of the pulmonary artery in mitral stenosis and right-sided hypertrophy is mentioned by Wilks and Moxon in their "Pathological Anatomy,' but modern text-books are mostly silent on the subject. Individual cases of pulmonary atheroma and endarteritis have been brought before the Society's notice by Drs. Wilks,[1] Bristowe,[2] Conway Evans,[3] and Sidney Coupland,[4] in which the naked-eye appearances of the larger and medium-sized branches have been particularly described. From his description, Dr. Conway Evans's case bears a very close resemblance to the one now under discussion. The stenosis, though much more marked than in this case, was evidently congenital, and there was considerable right-sided hypertrophy of the heart. He formulates from his case the following ingenious argument to explain the causation of

[1] This form of endarteritis is quite different from that striking variety which the writer has seen in syphilitic fibroid disease of the lungs, where the cell-proliferation is quite a predominant feature.

[2] Vol. xii. [3] Vol. xi. [4] Vol. xvii. [5] Vol. xxvi.

DESCRIPTION OF PLATE VI.

To illustrate Dr. G. F. Crooke's paper on Pulmonary Endarteritis. (Page 61.)

From drawings by Mr. E. Teichlemann.

FIG. 1.—A part of a larger branch of a pulmonary artery showing a nodose elevation of the intima. *a*. Consisting of hyaline fibroid swelling in which are embedded spindle-shaped cells. *b*. Tunica muscularis thickened. *c*. Patch of blood extravasation in *d*, the adventitia or periarterial fibrous sheath. Drawn under Hartnack, obj. 2, oc. 3. (35 diam.)

FIG. 2.—Longitudinal section of a medium-sized pulmonary artery.

a. Thickened intima consisting of a hyaline fibroid ground substance in which are contained branched (e^1) spindle- and round-cells undergoing fatty degeneration. e^2 and e^3.—Masses of cells fused together, undergoing fatty degeneration. *f*. Oil-globules, free in the ground substance, resulting from degenerated cells.

b. Elastic lamina.

c. Tunica muscularis.

d. Tunica adventitia.

Osmic acid preparation. Drawn under Hartnack, obj. 7, oc. 3. (270 diam.)

FIG. 3. A pulmonary arteriole affected with obliterative endarteritis.

a. Hyaline thickening of the intima almost obliterating the lumen.

b. Tunica muscularis, increase of muscle nuclei.

c. Adventitia infiltrated with small round-cells.

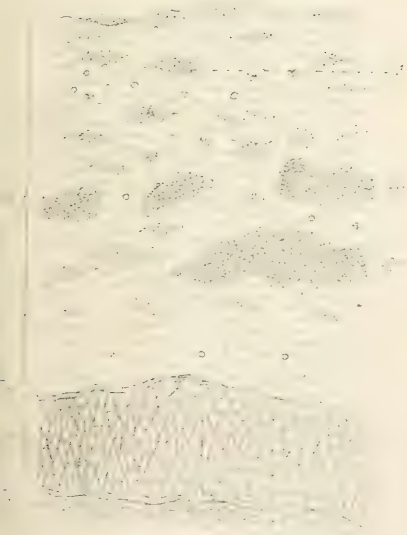
d. Dilated capillaries.

e. e. Pulmonary alveoli.

f. Small-celled infiltration around *g*, dilated and engorged vessels and capillaries.

h. Pigment masses (carbonaceous) in the plasm spaces of the perivascular connective tissue.

Drawn under Hartnack, obj. 4, oc. 3, tube half out. (100 diam.)



the lesion. First of all, in consequence of the mitral stenosis, a compensatory right-sided hypertrophy develops, which is sufficient up to a certain point to maintain the balance of the circulation. As the heart grows in accordance with the needs and requirements of the organism the equilibrium is disturbed, and as a consequence increased tension is thrown on the pulmonary circulation. Lastly, as effects of continuous and increased blood-pressure in the pulmonary artery, nutritive changes are set up in the walls, resulting in thickening and degeneration of the intima.

Now, even if this argument be regarded as not satisfactorily explaining pulmonary endarteritis in general, there is so much in it that is applicable to the case it is intended to illustrate, that I am constrained to adopt it in a great measure for my own.

Prof. Purser, of Dublin, has observed pulmonary endarteritis and atheroma in cases of mitral stenosis, and teaches that the continuously increased blood-pressure in the pulmonary artery stands in an intimately causal relation to the lesion. The only novelty I have to advance consists in the histological investigation of the lungs and pulmonary artery, which appears to show that the lesion begins at the periphery and spreads towards the large trunks of the artery. Secondly, as in most cases of brown induration of the lungs, so in this, there is to be observed a general thickening of the fibrous connective tissue in the lung, especially around the pulmonary artery and arterioles, where there is much small-celled infiltration to be occasionally noticed. Then also the extensive blood extravasation in the periarterial fibrous tissue around the larger branches, usually between the artery and bronchus, is another feature that should not escape notice. I would suggest, therefore, that the pulmonary endarteritis and atheroma in this case was produced by the combined effect of two causes, viz. (1) increased blood-pressure from within; (2) periarterial thickening[1] and infiltration from without, probably interfering with the proper nutrient supply to the vessel from its vasa vasorum.

In acquired stenosis of the mitral it must be admitted that actual endarteritis and atheroma of the pulmonary artery is very rarely observed. The writer has noticed in several cases general dilatation of the artery and yellow patches of simply fatty degeneration of the intima, but never, so far, those distinctly elevated nodes characteristic of endarteritis. *February 21st*, 1888.

[1] *Vide* paper on 'Endarteritis' by Dr. R. Saundby in 'Journ. Anat. & Phys.,' 1881

9. *Phthisical cavity with pneumothorax in an infant of six months.*

By J. A. Ormerod, M.D.

The patient, a female child aged 6 months, was admitted to St. Bartholomew's, care of Dr. Gee, for diarrhœa and marasmus. Five days afterwards it had a sudden attack in which its hands became contracted, it became blue, and gasped for breath. On examination of its chest no dulness was found; râles were heard all over the chest. It died seven hours later.

Post mortem, the right lung was found to be collapsed, and lying far from the chest wall close against the vertebral column. The pleura contained about half a drachm of thick pus, but this must have come from the perforation, as there was no sign of inflammation of the pleura. The lower lobe was simply collapsed. The middle lobe was solid, and externally presented a dark red spot in which was an opening the size of a crow-quill. A probe passed into the bronchus came out easily through this opening. On section a cavity was found about the size of a marble, with a fairly defined wall, on which was flaky pus. There was some reddish thick fluid in the cavity. Across it ran a whitish cord which on section was found to be a nearly obliterated vessel. This middle lobe contained grey tubercle, and also larger yellowish spots apparently filling the lobules. The upper lobe showed patchy emphysema and collapse. The bronchial glands of that side were enlarged and yellowish, and were found to contain tubercle bacilli.

The left lung was partially collapsed, but not nearly so completely as the right.

Except for one or two small spots of tubercle in the spleen, the remaining viscera were normal.

The mother of the child was phthisical.

January 3rd, 1888.

III. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Two cases of malformation of the heart.*

By H. Montague Murray, M.D.

Case 1. *Patent ductus arteriosus in a woman aged* 36, *dying of malignant endocarditis.*—M. S. H—, mantle maker. Delicate all her life, but never cyanosed. Had rheumatic fever ten years ago. Began to lose flesh March, 1887. Gave up work in May. Admitted into Charing Cross Hospital under Dr. Green in September.

On admission, heart was found to be hypertrophied and dilated. There was a systolic thrill over pulmonary cartilage, and a loud, harsh, grating systolic murmur to be heard all over cardiac area, but nowhere so loud as at pulmonary cartilage. Some of those who examined her maintained that a faint diastolic thrill and murmur could occasionally be made out over the pulmonary cartilage. There was some bronchial catarrh. Petechiæ soon appeared, especially in the extremities, together with hectic fever. Attacks of profuse temporary albuminuria and of temporary splenic enlargement followed. Patient rapidly wasted and died in December, 1887. There had been neither cyanosis or clubbing of finger ends.

Post-mortem examination.

Pericardium: Opaque patches scattered over ventricles and right auricle.

Right auricle normal.

Right ventricle: Recent vegetations on free borders of tricuspid valve. Pulmonary orifice dilated, valves large and competent. Oval mass of vegetations, $1\frac{1}{2} \times 1$ in., on the upper aspect of artery, beginning half an inch beyond attachment of valves. Just beyond the further end of this the pulmonary artery communicates with

the aorta by means of a passage $\frac{3}{8}$ in. long on the right side and considerably less on the left. The passage is, in shape, that of a short truncated cone, with base towards aorta. It just admits an ordinary quill. The wall of the aortic side is calcified.

Left auricle: Lining membrane roughened.

Left ventricle: Vegetations on attached borders of mitral valves and on free borders of aortic valves.

Interventricular septum: "Undefended space" thin and transparent. Weight of heart 18 oz. Hypertrophy general, but most marked on left side.

Liver fatty, cicatrised infarcts in spleen and kidney. Other organs normal.

This case presents two points of interest, the rarity of the malformation in those reaching adult life, and the apparent course of the blood stream.

Mr. Walsham has described a very similar case in the 'Transactions' of this Society (vol. xxviii), and at the same time referred to one reported by Dr. Fagge. The case resembles Mr. Walsham's in the absence of any excessive hypertrophy of the right ventricle, and Dr. Fagge's in the distinct dilatation at the commencement of the pulmonary artery. In Dr. Fagge's case there was marked hypertrophy of right ventricle. Regarding the course of the circulation, the evidence at our disposal points to the flow of blood having taken place from the aorta into the pulmonary artery (1), because the large mass of vegetations is situate in the pulmonary artery, and this would probably be formed in the eddy at the meeting-point of the streams; (2) because the pulmonary artery is dilated; (3) because there was no evidence during life that any venous blood entered the systemic circulation; and (4) because the murmur was heard most distinctly over the pulmonary cartilage. The absence of any great hypertrophy of the right ventricle shows that, at any rate before the onset of the malignant endocarditis, it had no great difficulty in emptying itself into the pulmonary artery, and probably, therefore, the intermingling of the two streams was slight. The condition of the heart does not suggest any cause for the patency of the ductus arteriosus.

Case 2. *Aorta arising from both ventricles; interventricular septum incomplete; stenosis of pulmonary artery; phthisis; tubercular meningitis.*—M. N—, a general servant, aged 26. No family

history of heart disease or phthisis. Excepting slight breathlessness, was quite well until twenty-one years of age. Then had rheumatism for six months. The exact nature of this attack, and whether it was accompanied by endocarditis, is uncertain; but since the attack she had never been well. During the five years that intervened between the rheumatism and her death, she suffered from frequent attacks of palpitation, bronchitis, cyanosis, œdema of feet, and paucity of urine. In one of the attacks it was observed that the cyanosis persisted after the other symptoms had been relieved. When she was twenty-four, periodic attacks of slight hæmoptysis, with cough and vomiting, set in, and clubbing of finger ends was noticed. At this time the abnormal physical signs were: a systolic thrill at apex, a systolic murmur at apex, conducted into axilla and heard at angle of scapula; also a systolic murmur at base, heard over aortic and pulmonary cartilages, up great vessels and down sternum. A soft diastolic murmur, following second sound, could also be distinguished. There were signs of commencing cavities at both apices. A year later, just before her death, the diastolic murmur was inaudible, but the other signs persisted. She was under the care of Dr. Mitchell Bruce at Charing Cross Hospital, where she died in January, 1888, with symptoms of meningitis.

Post-mortem examination.

Heart weighs 13 oz. Auricles and auriculo-ventricular orifices normal, except that there is a fine fringe of vegetations on valves of latter.

Aorta is seen to arise from both ventricles, though principally from left. Its commencement is three inches in circumference, but the valves, though large, are otherwise normal, and an inch from its origin the aorta is of ordinary calibre.

The interventricular septum has its concavity towards the left ventricle, though this is not so marked as in health, but at the upper part there is a crescentic deficiency corresponding to the origin of the aorta. The free upper edge of the septum is rounded and made up of muscle covered with endocardium.

The infundibulum is very small, but widens a little just before the pulmonary valves are reached. Here it suddenly terminates in a dome with a central opening in its summit, a quarter of an inch in diameter. The dome is composed of adherent pulmonary valves; outside it are three pouches, corresponding to the agglutinated

cusps. The rim of the orifice in the summit of the dome consists of much harder and thicker tissue than the remaining portion, and is uniformly fringed with minute vegetations. Beyond it the pulmonary artery is a trifle dilated but otherwise normal. The ventricular walls are of equal thickness. Both are thicker behind than in front, and at the base than at the apex.

Small, recent, phthisical cavities occupy both apices, and grey granulations and caseating nodules are scattered through remainder of lungs, which were of average size. Pleural surfaces are adherent.

Brain and membranes present usual characters of tubercular meningitis.

The features in this case to which I would draw attention are:

1. The age of the patient. I can only find one similar case in which the patient exceeded the age of twenty-six.

2. The apparent absence of cyanosis until the patient was twenty-one years of age. With such a degree of pulmonary stenosis, and with a second possible outlet for the blood in the right ventricle, it is difficult to understand how the blood could have been efficiently aerated, and I think, on the other hand, almost as difficult to believe that a girl of twenty should be cyanosed, and be unconscious of it.

3. How far were the defect in the septum and the stenosis of the pulmonary artery congenital? About the septum there can be no shadow of doubt. And the comparative frequency with which the two conditions are associated in infancy affords *a priori* evidence in favour of the congenital nature of the latter. In Dr. Peacock's book is a drawing that would almost represent the present case. But even in most of these it is impossible absolutely to exclude endocarditis. The safest hypothesis in this case is that there was some congenital stenosis, that upon it fell the brunt of the rheumatic endocarditis, and that thus the condition was exaggerated. The malformation is required to account for the smallness of the infundibulum, and for the regular conformation of the adhering valves, while the subsequent disease explains the onset and persistence of the cyanosis, and the fringe of vegetations round the opening. The cicatricial tissue in the margin of the dome may have been due to the force with which the blood was driven against it, but more likely to the endocarditis, which we know existed.

4. Then, lastly, there is the occurrence of tuberculosis in the

course of congenital heart disease. This I merely refer to as a case corroborating Dr. Percy Kidd's recent remarks in the 'St. Bartholomew's Hospital Reports.'

I have to thank Dr. Theodore Williams, Dr. Green, and Dr. Bruce for the use of the clinical notes of these cases.

May 1*st*, 1888.

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2. *Congenital malformation of heart and kidneys; obliteration of pulmonary artery; aorta arising from the right ventricle; imperfection of septum ventriculorum; lungs supplied from aorta by a large ductus arteriosus dividing into right and left pulmonary branches; horse-shoe kidney.* (*Card specimen.*)

By S. Herbert Habershon, M.D.

The subject of this case was a boy, aged 4 years, who first came under observation at the General Marylebone Dispensary in July, 1887. The child had been livid from its birth, and subject to frequent pulmonary attacks. The patient was small for his age, and presented the signs of extreme cyanosis, with great clubbing of the ends of fingers and toes. The mother had never suffered from rheumatism nor rheumatic fever. She states that at about the third or fourth month of her pregnancy she was attended from the dispensary for severe abdominal pain, accompanied by intensely painful and frequent micturition. She was said to have inflammation of the bladder, but I cannot obtain any record of the complaint or its treatment.

Physical signs.—Some bulging at the lower part of sternum and to the right of normal cardiac area. Forcible diffused cardiac impulse, chiefly on the right side of sternum. Apex-beat most plainly visible about three quarters of an inch below right nipple. Area of superficial cardiac dulness was more extensive to the right of the sternum than to the left, of about normal limits on the left side, but extending as far on the right as the right nipple, and upwards as high as the third costal cartilage half an inch from the right sternal border. Transverse dulness at this point one inch and nearly four inches at a line joining the nipples.

No bruit could be heard at any time, but the first cardiac sound was doubled and extremely forcible, and the second sound everywhere accentuated. Air entered feebly at the extreme bases of the lungs.

The patient improved for a time and continued fairly well until the autumn, when he contracted measles. He recovered from the attack, though he was very ill at the time. He continued under treatment through the winter. On March 27th he was suddenly seized with convulsions, and died comatose in the space of half an hour.

Post-mortem thirty hours after death.—Heart larger than natural for age of patient, and transverse diameter almost as large as vertical. Right auricle enormously distended, and filled with black fluid blood. *In situ*, only one vessel (the aorta) could be seen arising from a central position.

On opening the cavities of the heart, the right auricle is found to be dilated and thin walled, the vessels entering it normally, and the foramen ovale closed. A large coronary sinus opens into the auricle.

The right auriculo-ventricular valves are healthy and competent, but imperfectly divided into two unequal-sized curtains.

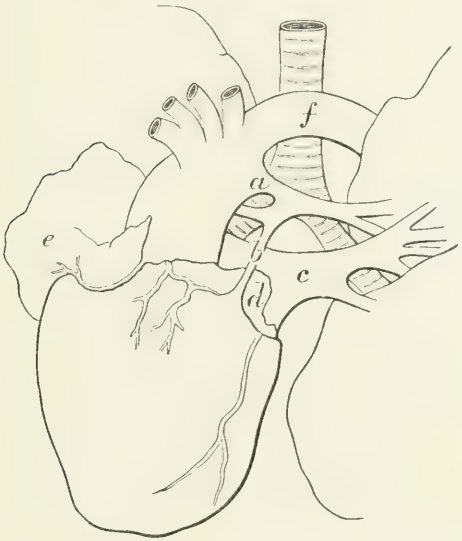
The right ventricle is enlarged and its walls much hypertrophied. The aorta arises directly from the right ventricle, and is of unusually large size. The sigmoid valves are very large and loose. Immediately below their attachment is an imperfection in the base of the septum of size sufficient to admit the little finger. The aorta must therefore during life have received blood directly from both ventricles. The left ventricle is of small size, its walls hypertrophied, but not to the same extent as the right. There is an opening into it from the left auricle through a bicuspid valve, normal in appearance, and a communication through the enclosed septum with the cavity of the right ventricle and with the aorta. No vessel opens from the left ventricle, and there is no trace of a pulmonary artery from within the ventricle.

The left auricle is atrophied and reduced to a venous sinus, into which the pulmonary veins open. The left appendix auriculæ is present, but very small.

The aorta is of unusually large size. Immediately beyond the point where the great vessels are given off it becomes suddenly constricted to about half its previous dimension. The lungs are

supplied from the aorta by means of a short trunk about a quarter of an inch in diameter, which is evidently the ductus arteriosus, and divides into a right and left pulmonary artery. Joining the right pulmonary branch is a vessel which can be traced to the heart to a point on the left of and a little anterior to the aorta. It is there lost in the muscular wall of the right ventricle. It is pervious near the pulmonary vessel and contains a little fluid blood, but near the heart it consists of a fibrous cord. The lungs are congested, but there is no collapse.

Woodcut 1.



a. Ductus arteriosus dividing into right and left pulmonary vessels. *b*. Pulmonary artery joining right pulmonary branch, and impervious near the heart. *c*. Thin-walled left auricle. *d*. Left auricular appendage (atrophied). *e*. Dilated right auricle (laid open). *f*. Descending aorta.

The liver and spleen are large, firm, and congested.

The kidneys are fused by a band uniting them at the lower part, presenting the usual horseshoe form of malformed kidney. On section the band is found to be composed of kidney substance, and not to be merely apparent. The right kidney is larger and extends

higher than the left. There is a separate hilus to each, directed backwards, and each kidney has its own ureter and vascular supply. The left suprarenal capsule is found to be nearly twice the size of the right.

Beyond this combination of malformations, which is rare, the only special point of peculiarity in this case (which, though not the commonest variety of malformation of heart, is by no means unusual) is the age to which the patient survived.

In commenting on a case of this kind, Dr. Peacock, in vol. xxii, p. 85, of this Society's 'Transactions,' remarks that the age of his patient (two and a quarter years) exceeded the period to which children with atresia of the pulmonary artery usually survive. In this case it will be observed that the child attained the age of four years. In a later case, however, and in several others recorded in his work on malformations of the heart, the ages of nine, ten, and thirteen years are mentioned as rarities. *May 1st*, 1888.

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3. *Fatty degeneration of the heart in cases of abdominal tumour.*

By Bedford Fenwick, M.D.

This specimen was removed by operation from a patient who was admitted into the Hospital for Women in August last. She was forty-years of age, had been married twenty-two years, and had had five children and one miscarriage. The abdomen had been considerably swollen for at least eighteen months before her admission. On examination, it was found uniformly distended by a solid mass up to the ensiform cartilage. The heart was displaced upwards, the apex beating on the level of the nipple, and the impulse being feeble and diffused. The cardiac sounds were very dull, especially the first sound over the right ventricle. The tumour was removed with considerable difficulty owing to its size, solidity, and extensive adhesions everywhere around. Except for vomiting and diarrhœa at first, the patient appeared to be doing well. The wound healed by the first intention, and the stitches were removed on the seventh day. But the pulse, which had been 90 before the operation, rose after it to 100, and then varied between 110 and

140. The temperature kept between 99° F. and 100° F., but this was attributed to the sultry weather. So matters went on till the eleventh day, when the patient became suddenly worse, and the pulse very rapid and feeble. She became unconscious about 7.30 a.m. on the twelfth day after the operation, and died suddenly about three hours later.

The *post-mortem* revealed a little peritonitis, chiefly in the neighbourhood of the wound. The various organs of the body were healthy, except the heart. The muscle of this was pale, yellowish, soft, and friable, the walls of the left cavities were of

Woodcut 2.

normal thickness, those of the right were very thinned, in places barely the thickness of cardboard. As the microscopic sections show, the muscle was in a marked state of fatty infiltration and fatty degeneration (Woodcut 2).

I venture to consider that this is a very important case. I have not the slightest desire to insist on the novelty of this subject, but I have searched with some little diligence through modern medical literature, and can find no notice anywhere of the matter. I would remind members of the Society that after operations for the removal of abdominal tumours, a certain number of patients die from peritonitis, septicæmia, hæmorrhage, &c. But there are a

No.	Social state.	Age.	Duration of symptoms and diagnosis.	Operation.	Date of death if after operation.	Cardiac condition.	Pulmonary state, &c.
1	M. $\frac{7}{1}$ (at 28)	39	Pain in r. side 3 years; swelling large for 20 months. *Ovarian cyst.*	R. ovariotomy	3rd day	L. ventricle large, r. very thin; fatty degeneration and infiltration generally.	Old pleural adhesions on the l. side; both lungs much compressed.
2	M. $\frac{5}{0}$ (at 26)	41	Doubtful, but of long duration. *Encephaloid of ovary.*	*Nil;* died very suddedly	—	Heart generally very soft and tissue fatty.	Old cavities at both apices; much phthisis in family.
3	M. $\frac{0}{0}$ (at 24)	44	Pain and swelling large for 9 months. *Ovarian cyst.*	Partial ovariotomy	40th day	L. ventricle large and soft: mitral valves thick; fatty degeneration, chiefly affecting r. heart.	Old adhesions of r. pleura.
4	M. $\frac{0}{1}$ (at 17)	37	Swelling large for 2 years. *Double ovarian cyst.*	„	3rd day	L. ventricle large, r. *very* thin walled; muscle very fatty.	Old adhesions.
5	Single	37	Much swelling 12 months; pain only last 6 months. *Ovarian cyst.*	L. ovariotomy	9th day	R. ventricle very thin walled and muscle very fatty; much fatty infiltration of l. side.	Much œdema of lungs.
6	M. $\frac{2}{0}$ (at 25)	43	Swelling 18 months. *Ovarian cyst.*	„	Fell down dead on 22nd day after operation	R. ventricle markedly thin; muscle generally very fatty.	Old pleuritic adhesions.
7	M. $\frac{1}{0}$	53	Swelling large only 5 months. *Ovarian cyst.*	Ovariotomy	3rd day	R. ventricle very thin; muscle generally very fatty.	Complete old adhesions all over r. pleura.
8	Single	40	Swelling ? 2 years. *Carcinoma of ovary.*	Exploratory incision	Died very suddenly 16 days after	R. ventricle very thin; muscle generally very fatty.	Secondary deposits in both lungs.
9	M. $\frac{0}{0}$ (at 27)	38	Swelling 14 months. *Ovarian cyst.*	Ovariotomy	5th day	Muscle generally very fatty, especially r. ventricle.	Lungs œdematous.
10	M. $\frac{4}{1}$ (at 26)	56	Swelling 6 years. *Ovarian cyst.*	Tapping	7th day, very suddenly	Muscle markedly fatty throughout.	L. lung carnified and adherent; r. pleura holding 20 oz. of fluid.

11	M. $\frac{3}{0}$ (at 26)	35	Swelling large for 7 months. *Ovarian cyst.*	Ovariotomy	2nd day	R. ventricle very thin and muscle very fatty; l. ventricle only infiltrated with fat cells.	Lungs deeply congested
12	M. $\frac{0}{1}$ (at 24)	32	Swelling large for 12 months. *Ovarian cyst.*	„	7th day	Muscle, especially of r. ventricle, very fatty.	Old calcareous nodules at l. apex.
13	Single	37	Swelling large for 13 months. *Ovarian cyst.*	„	15 hours after, never rallied	R. ventricle markedly thin and muscle degenerated generally.	R. pleura generally adherent; old calcareous nodules at r. apex.
14	M. $\frac{3}{0}$ (at 33)	65	Swelling 5 years. *Ovarian cyst.*	L. ovariotomy	2nd day, suddenly	R. ventricle very thin; muscle generally very fatty; aortic valves thick and calcareous; mitral valves less so.	4 oz. of blood-stained serum in each pleura.
15	Single	50	Swelling large for 7 months. *Ovarian cyst.*	*Nil*	—	R. ventricle very thin and muscle generally very fatty.	Pleuræ generally adherent.
16	M. $\frac{0}{0}$ (at 25)	44	Swelling 2 years. *Omental cyst.*	Tapped	6th day, suddenly	R. ventricle very thin and muscle very fatty.	Much emphysema.
17	M. $\frac{5}{2}$ (at 26)	42	Swelling 2 years. *Fibro-cyst of uterus.*	Exploratory incision	20th day	R. ventricle very thin; muscle generally very fatty.	Lungs deeply congested at bases.
18	Single	63	Swelling 2 years. *Fibro-cyst of uterus.*	*Nil*		R. ventricle very thin and muscle very fatty.	Lungs very œdematous and congested.
19	M. $\frac{0}{0}$ (at 22)	47	Swelling large for 4 years. *Fibro-cyst of uterus.*	Hysterectomy	3rd day; peritonitis	R. ventricle very thin and muscle very fatty.	Lungs very œdematous.
20	M. $\frac{2}{0}$ (at 31)	34	Swelling 12 months. *Renal cyst.*	*Nil*		Muscle generally very fatty; adherent pericardium; thickened mitral.	Pneumonia, l. lower lobe.
21	M. $\frac{3}{0}$ (at 21)	39	Swelling large for 2 years. *Renal cyst.*	„		R. ventricle very thin; muscle very fatty.	Both pleuræ thickened; old adhesions on l. side, recent on r.

few cases which rapidly sink after operation, and the *post-mortem* reveals no evident reason for death, and others which die without operation, nowadays, exactly as they died forty years ago, when Bright noted it, and wrote, "Some state of unexpected collapse occurs for which no reason can be assigned, and the patient suddenly dies" ('Lectures on Ovarian and Abdominal Tumours'). And even to the most recent writers who quote this remark of the great clinical teacher, this sudden death seems unexpected and inexplicable. Very deferentially, therefore, I desire to express an opinion that the cause of this rapid collapse or sudden death is cardiac failure from fatty degeneration of the muscular tissue of the organ. I have come to this conclusion only after six years' work at the subject, only after examining *post mortem*, twenty-two cases of abdominal tumour, which died more or less suddenly, with or without operation, and in every one of which this degenerative condition was well marked, and a tabular list of which I append. I am inclined to believe that the change is due directly to the pressure of the abdominal mass upwards on the diaphragm and thoracic viscera, because in about fifty other cases of large abdominal tumour which I have examined *post mortem*, and which died from other causes evidently than cardiac disease, I have frequently found the degenerative change beginning in the heart's muscle.

I am further led to think that the degree to which this degeneration progresses, depends partly on the patient's age, but chiefly on the duration and size of the intra-abdominal mass, and therefore to a large extent on the nature of the new growth, because it has been in cases of large cysts, ovarian, renal, or uterine, that I have noticed the degree of the degeneration to be most marked. As to the reason for the occurrence of the fatty change in the cardiac muscle it appears to me there is a simple physical explanation. For if we imagine the abdomen to be occupied by a large tumour, say cystic in nature, we know that as soon as the anterior wall of the cavity is distended by its growth there will be an equable pressure exerted upwards on the diaphragm, and as the cyst increases in size, so will that pressure increase in power. Therefore, the thoracic cavity will be more and more compressed, and thus with impeded pulmonary action the due oxygenation of the blood will be less and less properly performed. And this, it is well known, is a potent factor in the production of the fatty degenerative change in every part and organ of the body. If we add to this the

pressure, direct and indirect, to which the heart itself is subjected, the constriction upon its afferent and efferent and nutrient vessels, and add finally to this, the impairment of the general health, and of the power of locomotion of the patient, consequent on the progression of the disease, *prima facie* we might expect that muscular degeneration would occur. I would remind members of the Society how the muscles of the trunk and limbs wither and waste in these patients; how several and most accurate observers have noted that fatty disease occurs in the liver and kidney in these cases, and I would then contend for the inherent probability of the degenerative change affecting the cardiac muscle. Finally, I place these twenty-two cases of more or less "unexpected collapse" in every one of which the heart tissue was more or less extensively degenerated, and I trust that future observations, on a wider field, may prove whether or not these deaths are due to fatty degeneration of the heart. I must not, and need not, point out here, how important it is that the question should be solved as regards etiology, diagnosis, prognosis, and treatment. *December 6th*, 1887.

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4. *Cyst of heart.*

By W. B. Hadden, M.D.

The patient was a woman, aged 66, who died of cirrhosis of the liver.

During life the action of the heart was irregular, but there was no bruit.

There was nothing in the history which could explain the condition of the heart seen after death.

At the *post-mortem* examination a globular cyst, one inch and a half in diameter, was seen in the interauricular septum, projecting more into the right auricle than into the left. To the surface of the cyst the remains of the Eustachian valve were attached.

The walls, which were thin, were quite distinct from the endocardium and muscular tissue of the auricles.

After hardening in spirit, the contents were found to be pink,

grumous, unlike coagulum, and exhibiting no epithelial structures, hydatid membrane or hooklets.

Under the microscope the grumous material in the cyst was found to consist chiefly of minute granules and large granule-cells, which were shown by means of osmic acid to be fatty.

The wall of the cyst was composed of loose fibrous tissue, nowhere presenting the structure of skin.

There is no evidence that this cyst is either hydatid, dermoid, or serous. Its nature indeed seems quite obscure.

I can find no reference to cysts of this kind in the standard works on diseases of the heart or pathology. *April 3rd*, 1888.

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5. *Atheromatous sac on aortic flap of mitral valve, which is extremely stenosed.* (*Card specimen.*)

By G. N. Pitt, M.D.

The mass formed a soft closed sac projecting a quarter of an inch into the ventricle as a swelling half an inch by three eighths of an inch, situated about one eighth of an inch above the margin of the valve.

One end has been cut off to open the sac, which contained cholesterin and granular débris, but no pus-cells, although the material looked like thick pus with a faintly greenish tinge. On the auricular surface it scarcely projected, and adjacent to it were calcareous atheromatous nodules beneath the endocardium.

From Henry T— ; history of rheumatic fever at sixteen. Admitted after two weeks' illness with diarrhœa and pyrexia. There was a prolonged accentuated first apical sound. Pleuritic rub at right base; a trace of albumen in the urine. He developed thrombosis of left innominate and subscapular veins and died of cardiac failure.

Heart 17 oz. ; pericardium adherent ; mitral stenosis.

While fibroid and calcareous patches due to atheroma are common on the valves, I have not met with any record of a condition similar to that shown.

P.S.—The same evening Dr. Hadden showed a specimen of a closed cyst in the interauricular wall containing granular and fatty débris, which was probably a condition allied to that I showed.

April 3rd, 1888.

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6. *Mitral stenosis associated with pulmonary phthisis.* (*Card specimen.*)

By Percy Kidd, M.D.

The upper lobe of each lung presents an extreme degree of cirrhosis and contraction, the bronchi are dilated, and there are a few very small cavities. Another cavity of rather larger size is seen at the upper border of the left lower lobe with surrounding cirrhosis.

The lower parts of the lungs show a slight degree of brown induration, their anterior margins being somewhat emphysematous. A very few hard, miliary, pigmented nodules are seen scattered about the lower portion of each lung.

A scraping from one of the cavities contained numerous tubercle bacilli.

The heart, which weighs 13 oz., is considerably dilated.

The mitral orifice is contracted, of button-hole shape, and admits one finger. The valves are thickened and rigid, and a few flattish granulations are seen on their auricular surface.

History.—Patient was a male aged 25. Cough for nine years with several attacks of hæmoptysis. Scarlet fever in childhood, but never rheumatism. The patient, who was under observation for five years off and on, presented signs of double phthisis and mitral disease. He was admitted into the hospital about ten days before his death on account of hæmoptysis, which was followed by severe dyspnœa and signs of bronchitis from which he died.

April 17th, 1888.

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7. *Extreme aortic stenosis.* (*Card specimen.*)

By Frederick J. Smith, M.B., sent by R. Tuckett, Maidstone.

The specimen shows extreme aortic stenosis with great deposit of lime in the curtains of the valves. The aperture is reduced to a mere chink, and it would appear that only two curtains of the valve remain distinct. The other valves appear healthy and the aorta itself is remarkably free from disease.

The patient, aged 57, was a brewer's drayman, very powerful, accustomed to lifting heavy weights; never had rheumatism or gout, was perfectly healthy until six weeks before death, when he caught a chill, and pleurisy set in on the left side with much effusion.

A loud, harsh, rasping bruit, systolic in time, was heard all over the chest, but especially loud at the apex (which was displaced down and out) and over the middle of the sternum. He died shortly after admission to the Maidstone Hospital, chiefly through extreme dyspnœa. The left pleura was full of fluid and the lung collapsed, the right lung œdematous, all other organs quite healthy.

April 3rd, 1888.

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8. *Extreme stenosis of the orifices of the coronary arteries of the heart; sudden death.*

By F. Charlewood Turner, M.D.

The specimen is the heart of a man, aged 52, who was brought dead to the London Hospital, having been found in a dying state in a yard where he was working, about half an hour before.

The left ventricle is hypertrophied; its muscular tissue was of normal appearance, firm, and contracted. The first part of the aorta is very atheromatous and much dilated. The inner coat is much swelled, and the orifices of the coronary arteries have become extremely narrowed in consequence. The orifice of the left was found with some difficulty, and the end of a small probe passed through it. That of the right coronary artery could not be found

until the vessel was dissected up from behind. A small probe passed along the vessel was arrested at the orifice, but the conical end of a very small black French bougie was passed through the pin-hole aperture. Both coronary arteries were of good size up to the points of obstruction. The right was thin and collapsed, the left somewhat thickened and rigid. The curtains of the aortic valve are thickened along their attached borders.

The stomach contained much imperfectly masticated and undigested food. The viscera presented no important lesions. The brain was normal. The body was well developed and fairly nourished.

Stenosis of the orifices of the coronary arteries of the heart is recognised as a cause of angina pectoris, and of sudden death in that complaint independently of degeneration of the myocardium. It is perhaps less commonly a cause of sudden death in apparently healthy persons.

A case of this kind is recorded by Dr. Greenfield in vol. xxiv of the 'Transactions.' A woman, aged 27, was taken ill in the street and died in a cab on the way to St. Thomas's Hospital. And in 1884 I exhibited a similar specimen from a man, aged 49, who was taken ill in the street and died in a shop. Two cases are recorded by Dr. Ogle in the 'British and Foreign Medical and Chirurgical Review' for 1870.

In these cases there is no account given of the agonising pain of angina pectoris. And since it is probable that death occurs as a consequence of the lesion in the same way in all instances, the occurrence of such cases makes it doubtful whether the severity of the pain and mental distress is the direct or main cause of death in the typical anginose seizures.

Dr. Dickinson, in his remarks upon three fatal cases of angina pectoris referable to this lesion, attributes the symptoms to interference with the nutrition of the nervous structure of the heart, considering these the structures radically concerned in their production. And to interference with nerve nutrition also he would appear to attribute the fatal event, regarding the efficient nutrition, and, it may be, hypertrophic state, of the heart-muscle in these cases as evidence of the dependence of its nutrition, in greater or less degree, upon imbibition from the blood in the cavities.

I think, however, that the efficient maintenance of the nutrition of heart walls in the presence of an extreme degree of stenosis of

the orifices of the nutrient arteries, is a fact in accordance with, and an illustration of, the large provision made in the organization of the body to meet demands upon the functional capacity of different organs, under exceptional circumstances, far beyond the requirements of regular and tranquil life, and comparable in that respect with conditions of extreme stenosis of the cardiac orifices, or upper air passages found to be compatible with life.

Such a lesion of the coronary arteries, being of a chronic and more or less permanent nature, cannot be directly the cause of sudden death, but may bring about the result indirectly by interfering with the removal of exhausting products of muscular action, accumulated in excess in the myocardium by increased cardiac action due to the stimulus of muscular effort or mental excitement, just as stenosis of the mitral orifice, or of the upper air passages, may interfere with the removal of an excess of carbonic acid from the blood under similar circumstances.

Recovery from such a disturbance of the nutrition of the myocardium could only occur by depression of muscular action below the point for which the abnormal condition of the coronary circulation suffices, and the gradual restoration of the nutritive equilibrium.

May not the pain and sense of impending death of the anginose attacks due to this lesion, be the means by which the sufferer's life is preserved, when that is possible, by instantly arresting all movement and so affording the only chance of restoration of the nutritive equilibrium in the myocardium? And may not the fatal result in other cases be attributable to defect of such warning and paralysing sensations? *May 1st*, 1888.

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9. *Popliteal aneurysm; death from pulmonary embolism.* (*Card specimen.*)

By J. H. Targett, M.S.

History.—Richard S—, aged 39, clerk. A fortnight before admission, whilst walking was suddenly seized with pain behind right knee. A few days later the pain increased and a

swelling appeared. On admission, there was a large pulsating tumour in right popliteal space, which was diagnosed as an aneurysm. The leg was swollen and œdematous. Treatment by digital and instrumental compression at intervals during four or five days; which was followed by ulceration at the site of pressure. The patient progressed favourably till one morning he suddenly became faint, with difficulty in breathing, and died in a few minutes—seventeen days after admission.

Autopsy.—There was an embolus, doubled on itself, impacted in one of the main divisions of right pulmonary artery. No valvular disease. Thoracic aorta very atheromatous. No signs of syphilis. There was thrombosis of the femoral vein, commencing at the lower end of Hunter's canal and extending down the popliteal to the aneurysm. The vein was quite healthy opposite the seat of ulceration in Scarpa's triangle. Below the aneurysm the posterior tibial and peroneal veins were dilated, markedly varicose, and completely thrombosed. On section the aneurysm was found almost entirely filled with clot. Most probably the thrombosis commenced in the veins of the leg, and extended up the popliteal; and a portion of clot becoming detached formed the pulmonary embolus. There seemed no reason for connecting it in any way with the ulceration in the groin. *March 20th*, 1888.

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10. *A case of aneurysmal varix in a stump.*

By A. H. Robinson, M.D.

[With Plate VII, fig. 1]

The specimen shown this evening consists of a large portion of the popliteal artery and vein, and was removed during life from the stump of an amputation of the leg four inches below the knee-joint. It was removed by me some four years ago, the amputation having been performed sixteen years previously for gangrene following a severe crush of the foot and leg.

Upon examination it will be found that the popliteal artery, which has been laid open through its entire length, communicates at its distal extremity by a flap-like opening with the like

extremity of the popliteal vein. This, at least, was very apparent in the recent state, though the parts are somewhat altered now. Immediately after the arterio-venous communication, the vein dilates into the lower varicosity, and then, with the intervention of next to no trunk, again expands into the upper varicosity. Both varicosities are filled with recent clot, the ligature placed on the vessels at the time of operation having been left thereon.

The man from whom this specimen was removed is a muscular person, a turner by trade, and thirty-two years of age at the time of operation. He had always enjoyed good health, and has an excellent family history. After the amputation he wore an artificial leg of the ordinary box description, in which the knee rested flexed at a right angle, the stump projecting a very little behind. He never sustained any injury to the stump until the occasion on which I saw him, but he was in the habit of walking very long distances on the artificial leg, as much sometimes as thirty miles in a day. So he went on until some six weeks before I saw him in 1884, when he received a severe blow on the point of the stump. He suffered acutely at the time, but the pain subsided after a while. He became, however, unable to walk any distance, such exercise bringing about a recurrence of the pain. He then sought advice. When first seen, the posterior aspect of the stump was swollen and tense, while on placing the hand upon it, a diffuse and expansile pulsation was at once apparent. This pulsation was synchronous with that of the femoral artery, and ceased when the artery was compressed. No bruit could be heard over the swelling. There was no heat or redness of the skin. The man complained only of pain, and of this only since the accidental blow above mentioned.

As he stated that the swelling was becoming greater day by day, and was very anxious that something should be done, I placed a tourniquet on the thigh and cut down upon the swelling in the line of the popliteal vessels. I quickly came upon the lower varicosity, which I imagined to be an aneurysmal sac, and on dissecting round it the second varicosity appeared in the wound. A ligature was placed on the vessels as high up in the wound as possible, and the parts removed as now seen. The lower varicosity has been laid open to show the communication with the artery. The internal popliteal nerve is seen winding round the tumour.

October 18*th*, 1887.

DESCRIPTION OF PLATE VII.

Fig. 1 illustrates Dr. A. H. Robinson's specimen of Aneurysmal Varix in a Stump. (Page 85.)

Fig. 2 illustrates Mr. D'Arcy Power's case of Congenital Umbilical Hernia. (Page 108.)

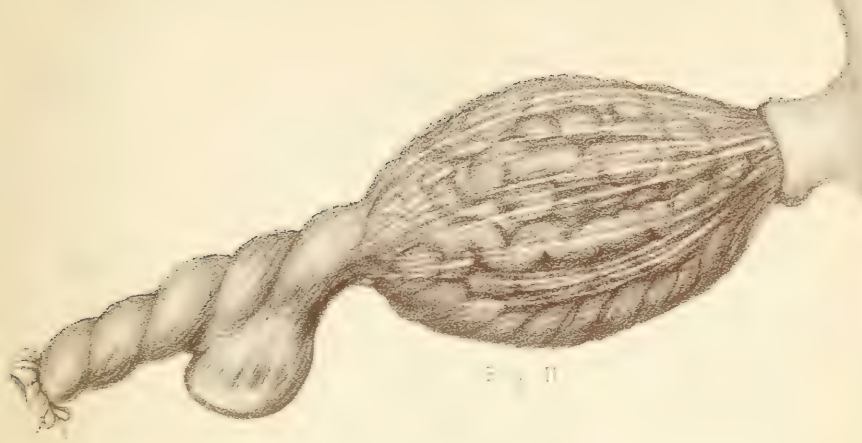

Fig. II

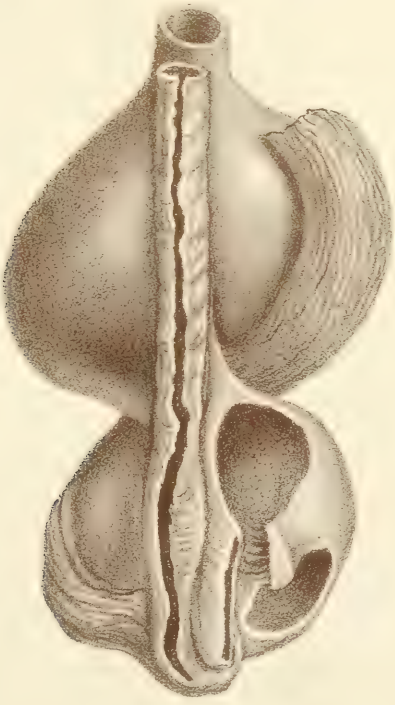

Fig. I

E. Burgess lith.

11. *Syphilitic arteritis from a case of general paralysis of the insane.* (*Card specimen.*)

By R. Percy Smith, M.D.

The specimen is a section of the basilar artery of a patient who died in Bethlem Royal Hospital, and shows well-marked thickening of the intima, agreeing very closely in appearance with a specimen figured in vol. xxxviii of the 'Transactions,' and shown last session by Dr. Sharkey. There is, however, rather more tendency to caseation and further some organised clot can be seen adherent to the inner coat on one side of the lumen of the artery. As in Dr. Sharkey's case the thickening is very irregular in distribution.

To the naked eye there was visible at the *post-mortem* examination, close to the junction of the two vertebral arteries, a round projection of the basilar artery of the size of a small pea, on section of which the coats of the vessel were found to be thickened, and the lumen was blocked by an old firm clot with a cavity in its centre. The rest of the basilar artery was occupied by recent soft black clot.

From a male, aged 34, who was first admitted to hospital in October, 1886, suffering from the early stage of general paralysis. He improved sufficiently to be discharged in March, 1887, and had a short period of remission of symptoms in which he was able to resume his occupation. He again broke down in June, became wildly maniacal, dirty, destructive, and erotic. On September 1st he was quieter when he suddenly became unconscious, and after some convulsive seizures became comatose and died.

Although a history of syphilis is common in cases of general paralysis of the insane, and in some would appear to be the determining cause of the mental and physical breakdown, yet coarse syphilitic lesions are rarely found *post mortem* in such cases, and therefore this case, in which a large cerebral artery was diseased, would seem to be of some interest, though it is not pretended that the symptoms observed during life were due to the particular lesions here shown. *December 20th*, 1887.

12. *Four cases of embolic gangrene.*

By Anthony A. Bowlby.

Case 1. *Multiple embolism of the vessels of the right arm with gangrene of the hand.*—J. B—, aged 63, a cabman, was admitted into St. Bartholomew's Hospital on November 25th, 1884, under the care of Mr. Savory, suffering from gangrene of the right thumb and part of the hand.

The patient stated that he had been a very healthy man until the last two years of his life, and denied any history of syphilis or drink. Had never had rheumatism. For the past two years he had been feeble and breathless, and unable to do any work. He said that a fortnight before his admission, whilst cleaning his boots, he was seized with sudden shooting pains in the thumb of the right hand, which almost at once became numb and cold. This condition was followed by swelling, and in a few days the thumb became black and lost all sensation.

On admission, the patient was found to be a well-built, tolerably healthy-looking man. The heart's action was very irregular and fluttering, the area of cardiac dulness increased, and both the first and second sounds were muffled and indistinct. Pulse in left wrist compressible, and artery tortuous.

The whole of the right hand was cold and numb. The thumb was black, shrivelled, and horny, the fingers blue, cold, and swollen, and all parts almost quite insensitive. No pulse in brachial, radial, and ulnar arteries. Very feeble pulsation in subclavian. The patient was kept in bed, the right arm was wrapped in cotton wool, opium was administered morning and evening, and a nourishing diet ordered.

The gangrene, however, slowly spread over the whole hand and extended into the wrist. The patient suffered much pain and slept badly. At times he was delirious. Temperature varied, but was generally above 100°, and the patient sank and died on December 27th.

A *post-mortem* examination showed that the heart was greatly enlarged and very fatty. The mitral orifice was much dilated, and the valve calcareous in patches. The aortic valves were atheroma-

tous and the aorta dilated. In the right axillary artery, at the point where the subscapular branch is given off, there was a large embolus, and below this the vessel was filled with clot for about an inch and a half or more; the clot was decolourised, firm, and adherent. The brachial artery was normal as far as the bend of the elbow, but was here filled by a large embolus, which had lodged at the bifurcation; the radial and ulnar arteries were each filled with clot for about half an inch.

Case 2. *Gangrene of leg from embolism of the right popliteal artery.*—M. C—, aged 55, was admitted into St. Bartholomew's Hospital under Dr. Gee on November 7th, 1884. She gave a history of an attack of partial right hemiplegia on the night of October 30th, followed by slight and transient aphasia. On October 31st there was sudden pain and numbness of the left leg. She had suffered an attack of rheumatic fever when eight years of age, and had recently had much shortness of breath.

On admission, the patient was found to be a feeble old woman with some cough and dyspnœa, and a little difficulty in articulation. There was no evidence of hemiplegia, but the sole of the left foot was gangrenous, and no pulsation could be felt in the arteries of the left lower extremity. The pulse at the wrist was good, on the left side feeble.

The heart's apex was displaced outwards, and to the left, and there was a loud presystolic apex murmur. After admission, the patient slowly improved, and the gangrene ceasing to spread, the dead tissue in the sole was gradually separated and cast off by November 30th. The pulse in the left femoral artery also returned, but none could be felt in the tibial vessels.

On December 12th, the patient was suddenly seized with severe pain in right leg and foot, generally followed by numbness and loss of colour in the limb below the knee. The foot quickly became cold, and no pulse could be felt below the femoral artery. The whole leg then became gangrenous, and on January 13th, amputation was performed by Mr. Willett in the lower third of the thigh. The sections of the popliteal artery showed that the vessel was filled with clot.

At 3 o'clock on January 13th, the morning after the operation, the patient had a sudden attack of right hemiplegia from which she never rallied, and gradually sank and died on January 15th.

A *post-mortem* examination showed that the mitral orifice was very much narrowed, and that the valve was extremely rigid. There were, however, no vegetations. The left auricle was greatly dilated, and the left ventricle hypertrophied. At the junction of the left middle, and anterior cerebral vessels was a large, recent embolus, and the left frontal convolutions were much softened. The remains of old emboli were found in the spleen and kidneys as well as in the popliteal artery above the stump of the amputated right leg. The left femoral and popliteal vessels were quite free from clot.

Case 3. *Gangrene of thigh and leg from embolism of right femoral artery.*—G. A—, aged 63, a policeman, was admitted into St. Bartholomew's Hospital on December 21st, 1885, with gangrene of part of the right thigh.

He stated that he had had rheumatic fever forty years previously, and that since then he had had occasional pain in the region of the breast. For a year he had suffered from dyspnœa and palpitation.

On the morning of December 9th he was suddenly seized with severe cramp-like pain in the left groin, followed by numbness and coldness of the whole limb. This was followed in ten days by gangrene.

On admission, the skin of the outer side of the left thigh was dry, black and gangrenous, and no pulse could be felt in the left femoral artery. The heart's action was very feeble, and accompanied by a loud systolic murmur.

On December 13th, gangrenous patches appeared over the leg and foot, and a slight pulsation was felt in the femoral at the level of Poupart's ligament, though not lower down.

On December 16th the patient sank and died.

After death the heart was found to be enlarged and fatty, and the aortic valves were covered with vegetations which were in front calcified. A large embolus plugged the common femoral at its bifurcation, and below this the superficial and deep femoral vessels were filled for three inches with clot which extended into the circumflex arteries.

Case 4.—*Gangrene of left foot from embolism of left femoral artery.*—M. J—, aged 37, a married woman, was admitted into St. Bartholomew's Hospital under the care of Mr. Morrant Baker on February 4th, 1888, with gangrene of the left foot.

The patient appeared to have enjoyed good health, and had led an active life. Ten weeks before admission she aborted, and nine days later, when still in a feeble state of health, she was suddenly seized with severe pain in the left groin whilst walking across her bedroom. This was quickly followed by burning pain in the foot and leg and numbness of these parts. The toes then gradually turned black.

On admission, the toes were dry, black, and shrivelled, in a typical condition of dry gangrene; the gangrene extended a little way on to the foot, where it was limited by a very definite line of demarcation. No pulse could be felt in the vessels of the left lower extremity. The heart's action was very feeble, but no murmur was detected.

On February 13th the gangrenous toes were removed, and the raw surface thus exposed gradually cicatrised, but had not completely healed when the patient left the hospital on April 11th.

The foregoing cases are very good examples of a variety of gangrene, of which but few instances have been brought before this Society, and they show very plainly the combination of conditions which by their association in cases of embolism bring about death of a limb. For, in cases of embolism the artery is not simply occluded as it might be by a ligature. On the contrary, an embolus by its lodgment at a bifurcation, occludes two vessels instead of one, and not only is the circulation thus cut off more effectually than in the case of a ligature, but the clotting around the embolus is more extensive than that which is found when an artery has been tied. Thus in Case 3, the clot occluded the superficial and deep femoral vessels, and extended into the circumflex arteries.

A second and most important factor in the causation of the gangrene is the lodgment of secondary emboli in the vessels beyond the seat of primary embolism. It is extremely well illustrated by Case 1, where the axillary, brachial, radial, and ulnar arteries were all plugged, and I have no doubt that smaller emboli, and more difficult to trace, are commonly present in most cases of embolic gangrene.

Lastly, attention must be directed towards the condition of the heart and great vessels. In all the cases here recorded, the heart was the seat of disease, and in most of them was in a very advanced state of degeneration; the arteries also were atheromatous. It is

evident that these conditions must greatly interfere with the development of a collateral circulation, for not only are the diseased vessels but little capable of enlarging to meet the altered requirements of the parts in which they are distributed, but the fatty and feebly-acting heart, which can only with difficulty pump blood to the extremities at the best of times, is quite unable to overcome the additional obstruction caused by the embolus, and cannot drive the blood stream along the narrow and tortuous channels of the collateral circulation.

Case 2 is also of interest on account of the evidence supplied that an embolus may be disintegrated. For there was a clear history of embolism of the left femoral artery followed by gangrene of the left foot; yet before gangrene of the right leg resulted from the lodgment of another embolus, the pulse had returned in the left femoral artery, and a *post-mortem* examination showed that the lumen of that vessel was clear. *April 17th*, 1888.

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13. *Two cases of gangrene caused by arterial thrombosis.*

By Anthony A. Bowlby.

Case 1. *Thrombosis of aorta, with clotting extending into the iliac vessels.*—E. B—, aged 56, was admitted into St. Bartholomew's Hospital under Sir Dyce Duckworth on September 23rd, 1885.

Just before her admission to the hospital, whilst walking home from work, she was suddenly seized with pain and numbness in the left leg.

On admission, the left leg was found to be pulseless and cold, and loud murmurs were heard over the præcordial region. The murmurs indicated disease of both the mitral and aortic valves. The patient quickly became very ill, and the heart's action very feeble. On September 28th the right leg became blue and cold, and on the 29th both legs were in a semi-gangrenous state. Death occurred on September 30th.

On *post-mortem* examination the heart was found to be enlarged, and both mitral and tricuspid valves thickened and atheromatous. The aorta was very atheromatous, and about three inches above its

bifurcation was filled by a firm dark clot which completely occluded its lumen, and extended into the iliac, femoral, and popliteal vessels on both sides.

Case 2.—S. F—, aged 48, a married woman, was admitted into St. Bartholomew's Hospital, under the care of Mr. Smith, on April 2nd, 1884, with dry gangrene of the left hand and forearm.

She stated that her general health had been fairly good all her life, though she had never been very robust. She had been married fifteen years, and had several times miscarried, but had never borne a living child. Her family history was good, her father being alive and aged seventy-five; several brothers and sisters were alive and well. She menstruated last in October, 1883.

With regard to her present illness, she stated that in the third week of December, 1883, she caught cold and had not been well since that time. She soon became very weak and feeble, and her legs, feet, and hands were liable to become cold, blue, and swollen.

In the first week of February, pain in the left hand became more severe, and the skin under the nail of the ring finger became black and shrivelled. The other fingers then became similarly affected, and very gradually the blackness extended over the whole hand and spread to the forearm. She suffered very severe pain of a burning character.

On admission, the patient was found to be a very thin and feeble woman, extremely anæmic, with dark circles under the eyes. Respiration was slow and very shallow. The pulse was extremely feeble, 90 to the minute. Heart's action similarly feeble, and sounds very faint, but no sign of any organic disease of the valves. Lungs natural. The legs were œdematous, but the pulse could be felt, although feeble, in all of the arteries. Urine normal. Bowels regular.

The left hand was in a typical condition of dry gangrene, being quite black, shrivelled, and mummified. The gangrene extended up the forearm in front as high as a point about two inches above the wrist, behind about four inches and a half above the wrist. No pulse could be felt in any of the arteries of the left upper extremity, but the brachial, axillary, and subclavian vessels felt as though they were distended with clot.

The patient suffered from severe burning pain generally referred to the hand.

A nourishing diet was ordered with small quantities of stimulants, and for a few days there seemed to be some slight improvement in the general state of the patient's health. This state of affairs did not, however, continue, and the woman became still more feeble, with failing appetite, much pain, and inability to sleep. The gangrene also slowly extended, but circulation in all other parts of the body, though very weak, did not further alter.

On April 10th, the patient was very apathetic and drowsy, answered questions slowly and with some confusion and hesitation, spoke thickly and with evident difficulty, and complained of pain in the head, and a sensation of giddiness. There was no paralysis. Pulse 120, regular, but very feeble.

On April 11th, speech was almost entirely lost and the voice was whispering. Pupils equal. Tongue protruded straight. General condition one of extreme lethargy and weakness.

From this condition the patient never rallied. The pulse became rapid, 150 in the minute; respiration was very shallow, the drowsiness deepened, and for twelve hours before her death on April 13th coma was almost complete.

A *post-mortem* examination showed that the brain was quite natural, and that there was no softening, thrombosis, or embolism. The lungs were collapsed at their base, and œdematous. The abdominal viscera were normal, except for a general shrunken and anæmic condition. The uterus was fixed by old perimetritic adhesions. All parts of the body were very bloodless. The heart was quite natural.

The whole of the arteries of the left upper extremity were filled with clot, which extended from the seat of gangrene to the origin of the subclavian from the aorta. The vertebral artery also contained some clot. There was a complete absence of all inflammatory thickening of, or around, the vessels, and no indication of an embolus in any artery. The veins were mostly empty. There was no thrombosis or plugging of any other vessel in the body. The structure of the plugged arteries was quite natural.

The gangrenous hand was removed, and, together with the heart and vessels, is now shown to members of the Society.

I have ventured to call these cases examples of gangrene from arterial thrombosis, but they differ widely from each other. Case 1 is an instance of a condition by no means rare, namely, clotting

of blood on an atheromatous patch, and it is only interesting on account of the very wide extent of the thrombosis, and the magnitude of the arteries thereby occluded. I think it probable that these results are in great measure due to the very advanced state of disease found in the heart. I do not suppose that they are to be accounted for by any condition of the vessel or the blood alone.

Case 2, however, is widely different, and as far as I can ascertain, unique. We have here to deal with a case in which apparently there has been a gradual clotting of blood in the vessels of a whole limb, without any antecedent embolism or arterial disease, and without any injury or inflammatory condition of the tissues of the limb or the affected vessels.

It is indeed very difficult to assign them to any recognised cause, but yet I think that the history of the case points rather to a general than to a local origin. I have described the extremely feeble and anæmic condition of the patient and the weakness of the heart's action, and would suggest that in this case there has been coagulation of the blood in the small arteries, thence spreading to the larger ones, as a result of excessive slowing of the arterial stream. In patients dying from exhausting diseases, it is of course common for clots to form in the veins, and that blood can clot in healthy arteries is shown by an examination of vessels after the application of ligatures. I have in several cases found extensive clotting far from the seat of ligature, though it would appear from an examination of vessels some time after operation that whole clots may be subsequently either partially or completely absorbed.

The history of Case 2 again differs very widely from the histories of those cases of gangrene due to embolism which I have already recorded. In this patient there was no sudden attack of pain and numbness, but, on the contrary, a clear history of coldness and numbness of all the extremities for at least a month before the gangrene commenced. The spread of the gangrene also was very slow. It would be impossible to exaggerate the excessive weakness exhibited by the patient, and the general feebleness of the circulation. *April 17th*, 1888.

14. *Carcinoma of the obliterated umbilical vein and hypogastric arteries.*

By W. Hale White, M.D.

Wm. A—, aged 48, was admitted for chronic constipation. There was a previous history of lead colic and gout. Four weeks ago he began to suffer from pain in the stomach and constipation. The latter has given rise to much difficulty lately, the motions being very hard, but no blood has been passed. There has been retching but no vomiting. On admission, he is a healthy-looking man somewhat wasted. In the abdominal wall, a little above the umbilicus and to the left of it, there is a hard rounded lump to be felt, the size of a filbert, and round the umbilicus there is a remarkably hard feeling of the abdominal wall, just as though the muscle of the rectus had been replaced by some solid growth. There is considerable pain in the abdomen, some vomiting, and obstinate constipation. The diagnosis was malignant disease of some organ with secondary growths in the abdominal wall. No direct evidence of the primary malignant disease could be found, although it was thought probable that it was in the abdomen. The patient died three weeks after admission. During the remainder of his life the emaciation was very rapid, the vomiting and constipation were both obstinate, and he gradually sank. The autopsy showed primary malignant disease of the pancreas, with secondary growths scattered about immediately under the peritoneum. But the point of especial interest which has induced me to bring the case before the members of the Society is that the two obliterated hypogastric arteries and the obliterated umbilical vein were converted into hard cords of malignant growth for almost the whole of their length. This was not due to spreading from neighbouring parts. This is a rare but very good example of the law that degenerate structures are particularly prone to be affected by malignant disease. The lumps to be felt during life were hard masses of secondary growth in the abdominal wall, which were sufficiently prominent to be felt from the outside. *January 3rd*, 1888.

IV. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. *Fibroma of the tongue.*

By Stephen Paget.

This little growth was removed from the tongue of a gentleman, aged 60. It had been noticed for four months, but had never given any pain. It was contained in the muscular substance of the front part of the right half of the tongue. It was about the size and shape of an olive; smooth, defined, rounded, elastic, and freely movable. The mucous membrane over it was movable and natural. It was shelled out with the greatest ease.

Its texture is that of loose wavy nucleated fibrous tissue; no gland-tissue is visible. *May 15th*, 1888.

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2. *Vascular tumours of the mouth and tongue.*

By Frederick Treves.

Case 1.—A gentleman, aged 57, consulted me with reference to severe bleeding from the tongue. Apart from the bleeding he was in perfect health, and was a hale, active, and vigorous man. He had had no serious illness in his life, was occasionally troubled with a little rheumatism, and was a member of a very rheumatic family.

He had since his childhood been the subject of epistaxis. This trouble was hereditary. His grandfather—who died at the age of eighty-one—had suffered from periodic bleeding from the nose all his life. His father, his two uncles, two of his brothers, one son

and two daughters, had always been troubled in the same way. There was no trace of hæmophilia in the family. The patient suffered from epistaxis very regularly every two to six weeks. He was liable to headaches, which commonly preceded the attacks and were invariably relieved by them. He was stout, and a trifle plethoric, but was the subject of no other obvious vascular trouble of any kind. He perspired always quite excessively. Four years ago he began for the first time in his life to bleed from the tongue. Since this form of hæmorrhage commenced he had never suffered from epistaxis. The bleeding from the tongue had been less frequent than that from the nose, and on one occasion he had lost no blood for a period of six months.

The hæmorrhage had become gradually more excessive in amount, and latterly had been so considerable as to cause faintness and some anæmia. The tongue was small, firm, and clean. Upon the dorsum were nine red tumours dotted irregularly about. The largest was placed about the centre of the tongue, and was the size of a split pea. It was soft, of a bright red colour, and disappeared entirely under pressure. All the other red spots were in front of this. The next in size had the dimensions of a hemp seed, while the remainder were about the size of pins' heads. The larger tumours had appeared first. The bleeding had been arterial. The blood had oozed from many of the points, but the gravest hæmorrhage had been from the largest of the tumours. Sometimes the bleeding had appeared quite spontaneously, at other times it had come on after food or while eating. It was evident that the raised specks were true angeiomata and were little masses made up of dilated arterioles and apparently bared of mucous membrane. I ordered the parts to be painted frequently with a 10 per-cent. solution of chromic acid, and in fourteen days all traces of the trouble had disappeared. The patient once more began to bleed from the nose, and this form of hæmorrhage has continued ever since upon the same lines as before. Into the causes of the bleeding it is impossible to enter. It was evident that for unknown reasons the mucous membrane of the tongue had taken the place of the mucous membrane of the nose as the seat of the periodic flux.

Baumgarten in his monograph upon epistaxis ('Die Epistaxis und ihre Behandlung,' Vienna, 1886) describes a precisely similar form of vascular growth upon the nasal mucous membrane in instances of repeated spontaneous nose bleeding. He speaks of

them as telangeiectic spots about the size of pins' heads that stand out as raised points upon the mucous membrane of the septum, or inferior turbinated bone. He adds that they may be demonstrated by a proper speculum. It is impossible not to note the connection between the headaches and their disappearance after a hæmorrhage.

Case 2.—A married woman, 31 years of age, came to the London Hospital in July, 1887, with severe bleeding from the tongue. She had been married eleven months, and had had one child born six weeks previous to her visit to the hospital. She appeared in sound health, but was a little flabby and anæmic. She was of fair complexion, and of good muscular development. She had had no illnesses of any note, but had suffered during the whole of her adult life from occasional attacks of violent frontal headache. During such attacks she was quite prostrated, and, as she expressed it, "was unable to lift her head up."

She had always lost much blood at each menstrual period. She had never had piles nor varicose veins, and had never bled from the nose. There was no fact of special moment in her family history.

The hæmorrhage of which she complained came from a solitary tumour placed upon the left edge of the tongue opposite the molar teeth. Apart from this the tongue was perfectly normal in appearance. The tumour was the size of a horse bean, was round, pedunculated, of bright red colour, and not unlike a raspberry in appearance. It had no visible pulsation, but could be reduced to a mere tag by pressure. It was painless. It had commenced to grow during the patient's pregnancy four months before she sought advice. It bled for the first time after the birth of the child, and it had since then bled frequently. The hæmorrhage was always spontaneous and had become latterly very alarming. On one occasion the patient maintained—in the rigorous language of a hospital patient—that "she had lost a quart." The bleeding was arterial. Since the hæmorrhage had been established the patient had had no headaches. I removed the tumour with scissors and restrained the free arterial bleeding with the actual cautery.

The tumour was soft, homogeneous, and of a faint pink colour on section. Under the microscope it showed itself to be a pure arterial angeioma.

Arterial angeiomata of the tongue would appear to be rare. Butlin—in his work on the tongue—makes no mention of any case of this character. He cites an instance of supposed arterial nævus and alludes to the comparative frequency of cirsoid aneurysms and of congenital venous nævi of the tongue. In the present instance—as in the last-named—a periodic headache was quite relieved by the loss of blood.

Case 3.—In connection with the example first recorded I may mention the case of another woman, aged 28, who presented a round pedunculated soft tumour the size of a hazel-nut upon the right edge of her tongue, in the precise position of the growth already described. It had existed for six months, had appeared during pregnancy, had never bled except when accidentally bitten, and had had no connection with headache. It was removed and proved to be a fibrocellular growth containing an unusual number of wide thin-walled vessels.

Case 4.—A woman, 29 years of age, was admitted into the London Hospital with a pulsating tumour growing from the periosteum of the right upper lateral incisor tooth.

The front teeth were perfect, but *all* the back teeth (molars and bicuspids) had been lost during pregnancies. The patient had had five children, and had always enjoyed good health. The tumour began two months ago as a small tag, felt with the tongue, and situated at the neck of the tooth, where it emerged from the gum. The patient was at the time four months advanced in pregnancy.

It grew rapidly and soon began to bleed. The blood that escaped was arterial. It now bled some four or five times a day, but no hæmorrhage had persisted for longer than five minutes. So free, however, was the bleeding while it existed that the woman had become anæmic. Her tongue was normal; the menstrual flow had never been excessive; she had never suffered from epistaxis, nor from any vascular trouble. She was the subject of severe prostrating headache, but she had had no attacks since the bleeding had commenced.

A soft, round, nodulated, bright red tumour, resembling in size, shape, and colour a raspberry, was fixed to the neck of the right lateral incisor tooth close to the gum. It presented very visible pulsation, could be almost made to disappear under pressure, and

was free from tenderness and pain. The central right incisor was quite loose. I removed this tooth and then extracted the lateral incisor. The tumour came away with it unbroken and without the least visible disturbance to its attachment. The bleeding from the socket of the tooth was arrested by a fine point of Paquelin's cautery.

The mass was obviously a pure arterial angeioma growing from the dental periosteum about the neck of the tooth. These tumours do not appear to be very uncommon. Salter, in his article upon the teeth in 'Holmes's System of Surgery,' writes as follows upon this subject:—"The tissues about the necks of the teeth are obnoxious to the growth of vascular tumours, which vary from passive nævus-like swellings to those other forms of more arterial character, aneurysms by anastomosis."

The author cites a case very similar to that just narrated, and states that these angeiomata are most often met with in adults in the upper jaw, and are either placed between the incisors or between those teeth and the canine.

In the last-named cases it is scarcely possible to avoid noting the connection between these growths and the conditions of pregnancy and of severe headache. *January 3rd*, 1888.

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3. *Ranula.* (*Card specimen.*)

By Frederic S. Eve.

A ranula showing small lobules of gland-tissue projecting from both its inner and outer surfaces. Microscopically these lobules of gland-tissue had the structure of tubular mucous glands. The contained fluid was very viscid, and gave no reaction to the tests for sulpho-cyanide of potassium. The submaxillary (Wharton's) duct was seen in dissecting out the cyst, but had no connection with it. The presence of gland-tissue in the wall of the cyst, its situation and contents, all correspond with the description of a ranula dissected *post mortem* by v. Recklinghausen, and believed by him to originate in the gland of Blandin and Nuhn at the

frænum of the tongue (see 'Virchow's Archive,' Bd. 84, S. 425). Von Recklinghausen concludes that this is the ordinary origin of ranula. *December 6th*, 1887.

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4. *Tubercular ulceration of tongue.* (*Card specimen.*)

By W. Hale White, M.D.

The patient, a man aged 39, gave fourteen months' history of rapid phthisis, with much hæmoptysis, sweating, and wasting. Six months ago he first noticed a pimple on the dorsum of the tongue. There are signs of phthisis over both lungs, and the larynx is very tender, and on examination is seen to be extensively ulcerated. The end of the tongue looks as though the anterior third had been removed by a transverse cut. The vertical surface thus presented is regularly indented by the lower teeth. The edge of this ulcerated surface is a little hard, and very slightly raised, whilst the surface itself, which is greyish red, is scattered all over with minute greyish granulations. The patient cannot give any date as to the first appearance of the ulceration at the tip. On the upper surface, extending backwards in the middle for an inch, and starting half an inch behind the tip, is a deep fissure, the edges of which are slightly hard and in contact. On separating them it can be seen that the surfaces of the cleft are covered all over with tubercles similar to those seen on the ulcer at the tip. Scrapings of these ulcers showed tubercle bacilli, but it was difficult to say for certain that they could not have come from some sputum that might have been in the mouth. There were no enlarged glands. The patient gradually sank and died. The *post mortem* showed phthisis of lungs, intestine, and larynx. The characteristics of the ulceration do not show at all well after death. *January 3rd*, 1888.

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5. *A submaxillary gland removed with an unusually large salivary calculus.* (*Card specimen.*)

By D'Arcy Power, M.B.

A submaxillary gland removed with a large submaxillary calculus. There is a deep depression at the upper and inner part of the gland where the calculus lay. At the bottom of this depression is Wharton's duct, cut across at the point where it leaves the gland. A piece of glass rod has been passed along its lumen into the gland-tissue. The gland itself is indurated, but otherwise appears to be healthy.

(*b*) The salivary calculus which was removed from the above gland. The stone weighs 4·4 grammes, and measures an inch in its long axis and one a half inches in circumference at its thickest part. It consists chiefly of phosphate and carbonate of calcium and magnesium.

(*c*) Cast of the preceding calculus to show its oval shape.

The gland and calculus were removed by Mr. Butlin from a gentleman aged 52 through an incision in the neck. The presence of the calculus was suspected, but was not discovered until after the removal of the gland. The patient had been subject to occasional swelling of the submaxillary region for more than forty years. There had been permanent enlargement and suppuration of the salivary gland for some time before the operation was performed.

The gland and calculus are preserved in the museum of St. Bartholomew's Hospital, Series xiv, No. 1826 (*a*) and liii, No. 245 (*a*). The cast is in Series lvi, No. 117 (*a*). *November 2nd*, 1887.

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6. *Dilatation of the œsophagus.*

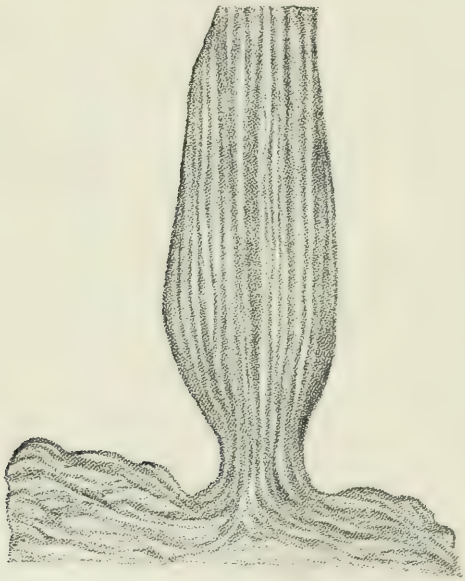
By H. Handford, M.D.

This specimen[1] was exhibited here as a card specimen in January of this year, forming one of a series of cases of œsophageal disease. It was taken from the body of a man, aged 52, who was

[1] The specimen is preserved in the Museum of the Royal College of Surgeons.

admitted under my care into the Nottingham General Hospital in July, 1886, complaining of shortness of breath, pain at the stomach, and vomiting. He had suffered from vomiting for five years at least. The so-called vomiting was found to be more truly regurgitation of food, and took place many times a day. Food was sometimes brought up at once, but often retained for half an hour or an hour before being regurgitated. There was no rumination as the regurgitated food was not chewed and swallowed again. He

Woodcut 3.



Œsophagus, with part of the cardiac end of stomach laid open. One fourth natural size.

had marked orthopnœa, systolic recession of the præcordia, and regurgitation in the jugulars, also a double aortic murmur and slight albuminuria. He died on the sixth day after admission.

At the inspection, twenty-four hours after death, the pericardium was found universally adherent, the heart much enlarged, and the cavities dilated. The aortic valves were incompetent, and the cusps thickened and calcareous. Weight of the heart with the arch of the aorta, $22\frac{3}{4}$ oz. The aorta was enormously dilated and the coats were atheromatous. The dilatation extended with

little alteration to the upper part of the thoracic aorta, after which it diminished rapidly, but when it passed through the diaphragm the aorta was still decidedly increased in size. The œsophagus was very markedly dilated throughout its whole length, except at its passage through the diaphragm, where it was contracted to very slightly less than the normal size. The dilatation was fusiform in shape, and the diameter about the middle of the tube, in the recent state and unopened, was just over two inches (Woodcut 3).

The œsophagus contained several ounces of fluid undigested food, chiefly milk and brandy. The walls were lax, and the tube, which appeared lengthened and was therefore curved, could be rolled like a sausage from side to side.

The measurements of the œsophagus when opened, and after being in strong spirit for five months, were :

Length	10 inches
Width, $2\frac{1}{2}$ inches from the upper extremity . .	3 ,,
Width, 4 ,, ,, . .	$3\frac{1}{3}$,,
Greatest width, $6\frac{1}{2}$,, ,, . .	$3\frac{1}{2}$,,
Width just above the cardiac orifice . . .	$1\frac{3}{4}$,,
Length of this narrowed portion . . .	1 ,,

The coats appeared thickened, but sections examined under the microscope and compared with sections of the walls of a normal œsophagus, showed no very great increase in thickness. The mucous and submucous coats were indurated and infiltrated with leucocytes, showing a chronic catarrhal change. No evidence of degeneration in the muscular coat was found.

The stomach contained only a little mucus, and there was no obstruction at the pylorus or other sign of disease. In the middle of the narrow portion of the œsophagus, just above the cardia, was a band of congestion about half an inch wide. It is probable that this was a *post-mortem* change.

There was no apparent cause for the obstruction of the œsophagus at its passage through the diaphragm, and the consequent dilatation above, unless, as seemed probable, the very greatly dilated aorta had caused some compression. *I think it quite possible that the dilated aorta may have to some degree pressed the œsophagus against the unyielding central tendon of the diaphragm and so interfered with the free passage of food.*

Another possible cause is general muscular atony, as in the cases

recorded by Klebs[1] and Luschka;[2] or disease of the vagus, for it has been shown[3] that after the vagus has been divided the food lodges in the lower part of the œsophagus.

I think the narrow part just above the cardia cannot fairly be regarded as a stricture. The narrowing is chiefly relative when compared with the dilatation above. The narrowest portion is one and three quarter inches wide after prolonged soaking in spirit, and during life therefore must have readily admitted the finger. There was no induration or thickening which could point to a cicatricial condition or new growth. Simple strictures, in addition to their excessive rarity, one would imagine to be almost of necessity congenital; and in this case no discomfort was experienced till nearly fifty years of age.

Simple dilatation of the œsophagus is rare, and Zenker,[4] in his article in 'Ziemssen's Cyclopædia,' gives the number of cases hitherto recorded as eighteen.

Luschka's case occurred in a woman, aged 50, who since her fifteenth year had been able to regurgitate her food without difficulty, and for some years before her death had been accustomed regularly to chew over again the regurgitated food. After death the œsophagus was found very greatly dilated. There was no trace of stricture, and the cardia of the stomach showed no departure from the normal. The œsophagus at its widest part was nearly as large as the forearm, and contained a large quantity of pultaceous material. The coats were thickened, the mucous less than the muscular; and Luschka considered it possible that the dilatation was caused by a severe catarrh of the mucous membrane, of which abundant traces remained.

Zenker's case forms a marked contrast to this in point of size. The breadth of the tube when split open (in an alcoholic preparation in which the tissues naturally were considerably shrunken) was 5 cm. at the greatest width in the middle of the dilated portion, while it measured only 3 cm. just below the pharynx and also just above the cardiac orifice. *October 18th*, 1887.

[1] 'Ziemssen's Cyclopædia,' vol. viii, p. 51.

[2] 'Virchow's Archiv,' Bd. 42, Taf. xi.

[3] Landois and Stirling, p. 328 (2nd edition).

[4] P. 52. Up to 1878. By Purton, Hannay, Rokitansky (2), Delle Chiage, Abercrombie, Oppolzer, Spengler, Wilks, Giesse (2), Ogle, Luschka, Klebs, Davy, Stern, Dare, and Zenker.

7. *Hypertrophy of circular muscular coat of œsophagus associated with an hypertrophied heart.* (*Card specimen.*)

By G. N. Pitt, M.D.

An œsophagus with marked hypertrophy of its circular coat which is over one sixth of an inch thick. The thickening is most marked an inch above the cardiac orifice, and extends as a very thick coat for five inches, and to a slighter degree for another two inches. The longitudinal fibres are not much hypertrophied.

From Louisa T—, aged 68, admitted under Dr. Goodhart with cerebral hæmorrhage and chronic Bright's disease. The kidneys showed also extensive acute changes in the convoluted tubes; the surface was granular and the capsules thick and adherent. Weight of kidneys 10½ oz.

Heart 21½ oz.; marked hypertrophy of left ventricle, and hypertrophy with dilatation of the right ventricle. Pericardial, pleural, and peritoneal adhesions.

No stricture, dilatation of stomach, or morbid growth could be found which would explain this muscular hypertrophy.

Wilks and Moxon noted the association of hypertrophy of the œsophagus with that of the heart, and this probably is the explanation of the present case, the pressure of the large heart on the œsophagus having incommoded the latter and caused difficulty in swallowing. *April 3rd*, 1888.

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8. *Œsophagus and stomach with numerous ulcers through which caseous glands have burst.* (*Card specimen.*)

By G. N. Pitt, M.D.

There are six groups of perforations connected with caseous lymphatic glands in different places along the œsophagus, the pus from each having burrowed through one or more openings with sharply defined edges.

The stomach shows several raised nodules in the submucous

tissue which contained pus. They vary in size from one twentieth to one sixth of an inch, are opaque white in colour, and on the lesser curvature near the cardiac end is a small perforation leading down to an adjacent caseous suppurating gland. There are two submucous tubercular nodules one tenth of an inch across in the jejunum.

From Mary S—, aged 67, admitted under Mr. Symonds with abscesses in the neck, on whom tracheotomy had to be performed on account of dyspnœa.

There was spinal caries affecting the sixth and seventh cervical vertebræ, and the pus, burrowing forwards, had set up caseation of numerous cervical glands. There were large caseous glands compressing the left bronchus, and to a less extent the lower end of the trachea. *April 3rd*, 1888.

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9. *A case of congenital umbilical hernia.*

By D'Arcy Power, M.B.

[With Plate VII, fig. 2.]

Although congenital umbilical hernia is not of extreme rarity, yet as no specimen has hitherto been exhibited before this Society, I thought that the following case was of sufficient interest to warrant my bringing it forward this evening.

I am indebted to the kindness of Mr. J. J. de Z. Marshall, of Lamberhurst, for the preparation, as well as for the particulars concerning it. "The mother is a healthy young woman, the mother of three previous children who were girls; the fourth, born on November 9th, 1886 was a fine full-term boy. The labour was quite natural, but a placenta-like mass came away with the abdomen. On closer examination by the aid of a candle this mass was found to be a transparent sac containing several coils of small intestine. It was situated in that part of the umbilical cord which was nearest to the abdomen of the child. The cord was ligatured and divided in the usual manner, and an ineffectual attempt was then made to replace the bowel through the umbilical opening.

After the application of gentle taxis for ten minutes, as the colour of the bowel became visibly deeper, the sac was carefully opened on a director; the gut, however, could not yet be returned into the abdominal cavity, and it was therefore found necessary to enlarge the umbilical ring. About a foot of small intestine was then with some difficulty passed into the peritoneal sac. The edges of the ring and of the bowel were subsequently brought together with silver wire, carbolised dressings were applied, and the abdomen was supported with a broad strip of plaster and a bandage. The child survived his birth and the operation for three days, dying of peritonitis.

The tumour appears to be formed by a dilatation of the coverings of the proximal portion of the funis. It is fusiform in shape, and has the main constituents of the umbilical cord running as a bundle along its lower border. The wall of the sac consists of a thin and soft membrane, which was so transparent whilst the specimen was fresh that the coils of intestine could readily be seen through it. The external surface was polished, and closely resembled the outer surface of the cord, whilst internally the sac is covered with a smooth layer, which is apparently derived from the peritoneum. At the apex of the tumour the funis reappears, and has upon its under surface a cyst containing a viscid fluid. A bristle has been passed for a short distance into the umbilical vein, and a green rod has been passed between the sac wall and its peritoneal lining.

I find that Antonio Scarpa and Sir William Lawrence, in their classical treatises on rupture, have given a complete account of this variety of hernia, and their descriptions apply in nearly every respect to that which I have just read. The present case is only remarkable in that the child had no other imperfection, for both these authors state that it is often associated with some deformity, usually spina bifida, and on the presence of the second smaller cyst filled with fluid.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series xx, No. 2156 (*a*), and a drawing made before the return of the rupture in Series lvii, No. 260. j.

October 18*th*, 1887.

10. *Adenomatous polypus of umbilicus.* (*Card specimen.*)

By W. S. Colman, M.B.

The polypus, the size of a split pea, distinctly pedunculated, was removed from just within the dimple of the umbilicus of a child, aged two months. It was first noticed when the child was a fortnight old. The umbilicus was closed.

Microscopically, the polypus was composed of a core of ordinary unstriped muscle, and covered by a thick layer of mucous membrane, which contained Lieberkühn's follicles, adenoid tissue, &c., exactly like the normal mucous membrane of the small intestine.

May 15th, 1888.

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11. *Volvulus of the cæcum.* (*Card specimen.*)

By W. J. Walsham.

The specimen consists of the transverse colon, the ascending colon, the cæcum, a few inches of the ileum, and a portion of the abdominal parietes.

The colon, tracing it backwards from the splenic flexure, instead of passing transversely across the abdomen, descended to the left iliac fossa and thence turned upon itself and ran up again to the stomach, forming the U-shaped bend seen to the right of the specimen. From the stomach it passed nearly vertically downwards to the cæcum, which was situated over the last lumbar vertebra almost in the middle line. Thus there was no ascending colon in the normal situation in the right loin.

The cæcum and the beginning of the colon are twisted three times from right to left around the lower few inches of the ileum, forming a corkscrew-like coil with the cæcum at the apex. The piece of the abdominal parietes to which the cæcum is firmly attached by sutures was removed from the middle line of the body midway between the umbilicus and the pubes, the spot at which the abdomen was opened during life for the purpose of relieving

the intestinal obstruction from which the patient was suffering. The cæcum was the first distended coil that presented at the operation, and was at once secured to the abdominal parietes and opened. No exploration of the abdominal cavity was made, as the patient's condition at the time of the operation was too desperate to admit of it. The securing of the cæcum—the apex of the volvulus—to the abdominal wall effectually prevented the untwisting of the volvulus. There was general peritonitis, and the cæcum in places was gangrenous, but there was no escape of fæces on account of surrounding adhesions.

The notes of the case were read at a recent meeting of the Clinical Society. *February 21st*, 1888.

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12. *Anchylostomum duodenale; intestine and parasite from a patient infected with the above.*

By W. Hale White, M.D., for Dr. Rake, of Trinidad.

B., aged 40, Hindu, admitted into the Trinidad Leper Asylum, June 14th, 1887, died November 16th, 1887. Tuberculated leprosy of three years' duration.

Chief symptoms during life:

1. Pyrexia (highest temperature 104·4°) partly due to outbreak of tubercles.
2. Intense progressive anæmia.
3. Œdema of hands, with no albumen in the urine.

Necropsy showed the right pleura to be full of fluid; there was also some in the left pleura and peritoneum. Miliary tubercles in the lungs, liver, and spleen. Anæmia of kidneys. Swarms of *Anchylostomum duodenale* with hæmorrhages in the upper part of the jejunum. None were found in the stomach, duodenum, or large intestine. The ileum contained much black, disintegrated blood. The brain, heart, and testes were healthy. Some fasciculi of the median nerve were thickened. Magenta showed numerous leprosy bacilli in the median nerve and femoral gland, a few in the testes, none in other viscera. *January 3rd*, 1888.

13. *Tubercular ulceration of the intestine, associated with the presence of fruit-stones.* (*Card specimen.*)

By Leopold Hudson.

This specimen was obtained from a boy, aged 12, who had been under the care of Dr. Finlay in the Middlesex Hospital. The ulceration involved the cæcum and the lower six feet of small intestine. The entire mucous membrane of the cæcum had been destroyed, and it had undergone much contraction ; its walls were infiltrated with caseating tubercular deposit, being four times their normal thickness ; the margins of the ulcer were scalloped, thickened and overhanging, and to the surface were attached filamentous shreddy sloughs. Lying in the cæcum were found many plum-stones and cherry-stones. The ulcers in the ileum were numerous and of typical tubercular character ; but, contrary to the general rule, those highest up were more extensive and deeper than those nearest the ileo-cæcal valve. The ulcers in the middle of the ileum completely encircled the gut ; they exposed the muscular coat and presented, from irregular extension, a honeycombed appearance, some islets of mucous membrane escaping the destructive process. In many of them contraction had taken place, reducing the calibre of the gut quite one half, the peritoneal aspect being thickened, fibrous, and showing numerous tubercles beneath the surface. Lying in the lumen of the gut opposite to each of these contracting ulcers one, two, or three fruit-stones were found, which were of very dark colour, and presented a rough surface. Lower down, the ulcers, though more numerous, were more superficial and of irregular shape ; the lowest twelve inches of the ileum presented deep punctate pigmentation.

Many of the mesenteric glands were enlarged and caseous, the spleen was lardaceous, and there was recent tubercular deposit in the left lung. Microscopical examination confirmed the view that the intestinal lesions were tubercular. A question of great interest raised by this specimen was to what extent the presence of foreign bodies had been instrumental in determining the extent and position, and even indeed the origin of the ulcers. *March 6th*, 1888.

14. *Stricture of the intestine following tubercular ulcer.*

By Frederick Treves.

The following is a case of primary tubercular ulcer of the bowel that led to a fatal stenosis.

The patient, a lad of 15, was admitted into the London Hospital, under the care of Dr. Stephen Mackenzie, on March 7th, 1887. He was suffering from symptoms of chronic intestinal obstruction. He was sickly and ill developed, and a member of a phthisical family. The abdominal trouble had commenced some six months before admission with diarrhœa, followed by a gradually increasing constipation with colic, and later on with nausea and sickness.

During the patient's stay in the medical wards some changes were noticed in the apex of the left lung, the lungs having been reported as normal on admission.

The symptoms of obstruction became more and more marked, and the patient rapidly lost flesh and strength. Finally a comparatively acute attack supervened, for which I performed enterotomy. The patient's condition was desperate at the time, and peritonitis had already supervened. He died a few hours after the ileum had been opened, and about one month after his admission into the hospital.

The autopsy revealed general peritonitis. There had been no extravasation of fæcal matter. The artificial anus was in the lower end of the ileum. This part of the small intestine was enormously dilated, and its walls were greatly thickened. The cæcum, commencement of the colon, and terminal part of the ileum, were adherent to the iliac fossa. The cæcum was contracted, and behind it lay a short and adherent appendix. A stricture occupied the bowel at the point where the cæcum and ascending colon joined. The stricture formed a hard, rigid, circular ring that would just admit the tip of the little finger. Two enlarged caseous glands were found in the vicinity of the stricture. The mesenteric glands were a little more conspicuous than normal. No tubercles were to be found upon the surface of the peritoneum.

Two superficial ulcers, probably stercoral ulcers, occupied the ileum. The rest of the alimentary canal was normal.

Caseating deposits were found in the apex of the left lung. With this exception the remaining viscera exhibited no morbid change.

The stricture was circular, and composed of very dense material. The cicatrix was limited to the walls of the bowel, there being very little thickening of the tissues outside the tube. The narrowed area occupied about an inch of the intestine measured in its long axis. There was no ulceration present. The mucous membrane had been converted into a dense cicatricial tissue. The edges of the scar were clearly defined from the adjacent normal mucous membrane, were much raised and well rounded. The greater part of the membrane was entirely undermined and separated from the muscular coat. It formed a very firm dense bridge of tissue, fenestrated with cloacæ in some twenty places. These cloacæ had smooth rounded margins, and led into an even-walled submucous cavity.

From every part of the surface of this fenestrated scar hung a series of remarkable tags of tissue, about fifty in number. Some were short and sessile, others reached to three quarters of an inch in length, and were club-like or ended in rounded branched processes. They were evidently formed from shreds of mucous membrane that had been dissected off by a destructive process and had been converted into tough cicatricial appendages. It was clear that the stenosis was mainly due to the contraction of the mucous membrane. It would appear as if the ulceration had commenced in a deposit in the submucous tissue that had subsequently broken down and had undermined and partially destroyed the mucous membrane. The situation of the ileo-cæcal valve was indicated, but it had been widely opened up by the enormous dilatation of the ileum.

The case may be taken as an example of primary tubercular ulcer of the intestine leading to stenosis of the canal, and associated with subsequent tubercular changes in the lung.

Tubercular ulcers of the intestine are met with frequently enough in the *post-mortem* room, but I imagine it is very rare to find that these ulcers have led to a definite stricture of the bowel. The term "tubercular" is given to these lesions because they are found associated with tubercular manifestations elsewhere, with phthisis, or with other and more widely spread forms of tuberculosis. The ulcer has certain precise local characteristics, but it is

questionable whether these are always sufficiently emphatic to alone form the basis of a diagnosis. The diagnosis is made, indeed, as much from the environment of the sore as from its special appearance.

The trouble is usually secondary, and appears, in the great majority of cases, as a not very important element in an important disease.

On the other hand, it is clear that the tubercular ulcer of the bowel may appear as a primary affection, and that even in the midst of other tubercular manifestations it may hold—for a time at least—the position of the predominant disease.

It may be met with in any part of the bowel, but it especially selects what may be termed the typhoid region. It is in fact most common in an area of the bowel of which the ileo-cæcal valve is the centre, especially attacking the lower extremity of the ileum, and less frequently the cæcum and the commencement of the colon.

The ulcer begins in the lymphoid tissue, in the solitary glands, and in Peyer's patches. It first takes the form of a grey, non-vascular speck that stands out from the surrounding mucous membrane. This island grows. If many appear in Peyer's patches they may define the peculiar oblong outline of those districts. In other parts the affected spot remains rounded. This stage is well illustrated by a specimen in the College of Surgeons Museum taken from the bowel of a Cariama, Nos. 2546, 2547. The intestine in the specimen is injected, and the affected spots stand out boldly as little pale nodules against the bright red mucous membrane. The deposit soon breaks down by ulceration, and then the diseased area sinks below the level of the surrounding surface. The ulcer spreads. It takes an oval outline, and nearly always the long axis of the patch is parallel with the transverse axis of the gut. The sore indeed tends to spread right round the lumen of the bowel, and to form at last a complete ring of ulceration. Often the ulcer will be found to have extended along one half or three quarters of the circumference of the tube.

The ulcers are nearly always multiple, and as they vary considerably in size in the same specimen it is evident that they differ in point of age, that they appear in succession and not in a simultaneous outbreak.

The ulcer soon becomes excavated. Its edges are flat, ragged, and sharply cut, thinned and peculiarly disposed to become in time

undermined. In the earliest stage this undermining is not apparent. As the case progresses it may become very extensive, and give to the ulcer a characteristic appearance. The general outline of the lesion is regular. The base is very uneven. It may at first appear occupied by a caseous-looking deposit, but usually it is covered by exuberant and irregular granulations, that may rise above the normal level of the mucous membrane. In a later stage the base becomes more even and possibly quite smooth, and may then exhibit no active inflammatory process.

The undermined margin of the ulcer is apt to become unevenly destroyed, and there result peculiar tags of mucous membrane that fringe the sore. Some of these may attain an inch in length. If the ulcer heals or heals in part these tags persist as elongated or rounded or club-shaped processes. They are well seen in the specimen exhibited. In other instances bridges of mucous membrane are left, or cloaca-like apertures appear in the undermined and widely separated membrane.

In cases where the destructive process advances rapidly the undermined edge may become broken up into fine flocculent shreds. A specimen in the College of Surgeons Museum well illustrates this.

If the ulcer heals the margin becomes raised, rounded, and smooth. It appears turned in, and beneath it is often a large submucous space, due to separation of the mucous membrane. A good example of an ulcer in process of healing is afforded by Specimen No. 2545 in the College collection.

In the nooks and recesses formed about these ulcers caseous masses may sometimes be found.

As already stated, it is exceedingly unusual for these ulcers to lead to a definite stricture of the bowel. They may lead to a little narrowing of the lumen of the tube, and specimens of this condition are not uncommon. In my work on Intestinal Obstruction I have given a figure of a stricture of the jejunum, said to be due to tubercular ulceration. The specimen is in the London Hospital Museum, and appears rather to be an example of multiple simple stricture of the bowel than of cicatrix due to tubercular disease.

With the exception of this doubtful specimen, I am not aware of any example in the Metropolitan museums of a stricture of the character now under notice.

The very nature of the tubercular process and the usual progress

of the trouble are strongly opposed to the formation of cicatricial tissue. In scrofulous and tubercular lesions elsewhere that have undergone healing the amount of cicatricial tissue developed is usually of the scantiest. To produce a stricture of the bowel, and especially a stricture of the colon capable of causing symptoms, it is necessary that a large amount of contracting fibrous tissue should be forthcoming. The scrofulous and tubercular ulcer has singularly little disposition to form this material, and thus it is that a permanent stenosis is so uncommon.

With this ulcer of the intestine some enlargement of the mesenteric and meso-colic glands is invariable. Such glands may form large masses, and they are apt to persist even after the ulcers have healed. The condition of the glands is an important element in the diagnosis of the lesion.

In the advanced cases that commonly present themselves in the *post-mortem* room it is not unusual to find a deposit of tubercles throughout the peritoneum, and it will often be observed that such tubercles are most numerous in the immediate vicinity of the ulcerated bowel. *December 6th*, 1887.

15. *Multiple tubercular strictures of the intestine.* (*Card specimen.*)

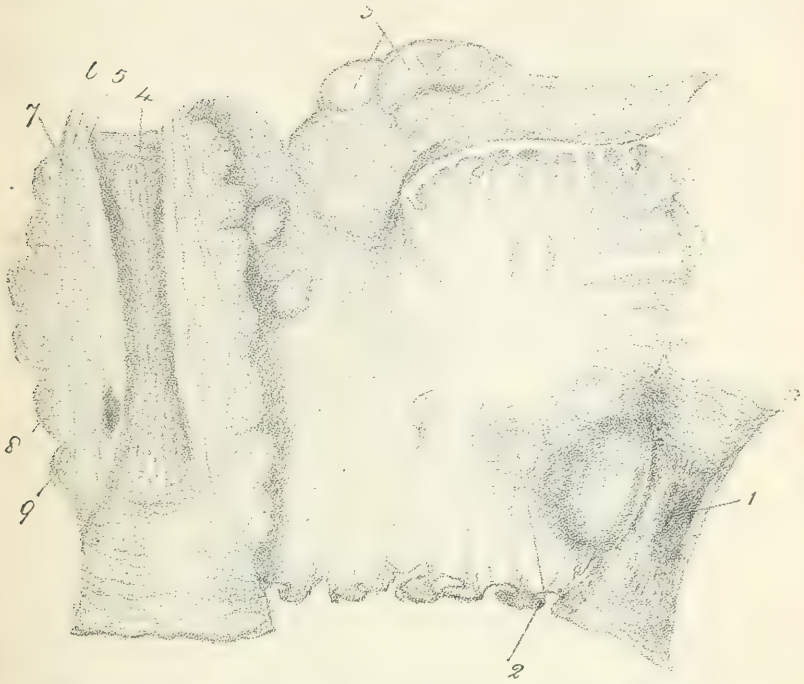
By H. Handford, M.D.

The specimen was taken from a lad aged 17. He was pale and pasty looking, rather thin, and had a resistant, distended abdomen, but was moderately well developed. He had two calculi removed from the bladder when nine years of age. Otherwise he had been fairly well. His father and mother were alive and well; the history of brothers and sisters, &c., was very indefinite. He had been out of work, and consequently short of food, for five months. His illness began four months before his admission (January 25th, 1888) with the passage of small quantities of bright red blood *per anum* on two or three occasions. He subsequently had irregular diarrhœa and wasting, and lost strength very much. He had very little cough and no sputum, but there were signs of

some consolidation at both apices, and dulness in the interscapular region, with tubular breathing, which led to a suspicion of enlarged bronchial glands. The cervical and submaxillary lymphatic glands were greatly enlarged on the left side. No enlargement of inguinal or axillary glands. His temperature varied between 97·4° and 101·8° at first, but later kept much more uniformly about 99°. The bowels were moved from two to four times daily, and were but little influenced by a variety of astringents, Nevertheless he gained 8 lbs. in weight. Eventually he began to pass bright blood frequently with the motions, and complained of pain at the "seat" in defæcation. The stools were noticed to be formed, but small, *i. e.* cylinders about half an inch in diameter. On rectal examination a very tight stricture was felt just within reach of the index finger. It was very painful to the touch and was surrounded by an indurated mass as large as a small orange. A gum elastic rectal bougie, about the size of a No. 20 catheter, was passed, under chloroform, by my colleague Mr. Wright. The patient continued as usual till twenty six hours later, when the bowels were moved, and he immediately complained of violent pain. He died thirty-eight hours subsequently of peritonitis.

Inspection fifty-six and a half hours post mortem.—Rigor mortis still present. Hypostatic congestion moderate and of a bright red colour. Abdomen moderately distended. Intestines reddened and glued together by recent lymph. An area just above the bladder was shut off from the rest of the peritoneal cavity by adhesions and was filled with pus. There was no sign of fæculent extravasation. The small intestines were moderately distended with flatus; the greater part of the large intestine was collapsed. There was about a pint of semi-purulent fluid in the abdominal cavity. The pelvis was occupied by a large, ill-defined mass, which could only be removed by free cutting. On the peritoneal surface of the upper portion of the rectum was an opening, irregularly rounded in shape, and about 5 mm. in diameter, where the bougie had passed through the walls of the intestine. The rectum and sigmoid flexure were removed together with the surrounding mass, which consisted chiefly of fat, indurated by excess of fibrous tissue, and having several enlarged glands embedded in it. This fatty growth completely encircled the intestine, and was continuous with its walls. When split open the thickness of the fatty growth was 3—5 cm. on each side. The thickness of the intestinal wall in the stricture

Woodcut 4.



Mesentery, with greatly enlarged glands, pancreas, and two portions of small intestine; the left-hand one embedded in a mass of indurated adipose tissue continuous with, and growing from, the mesentery. Both pieces of intestine are narrowed by stricture; they have been laid open by longitudinal incision, and the margins turned back. From a sketch by Dr. Handford. Half natural size.

1. Deep ulcer in constricted portion of small intestine (lower end of jejunum or beginning of ileum) leading into an enlarged glandular inflammatory mass (No. 2), which has encroached upon and involved the wall of the intestine.
2. Inflammatory tubercular mass originating in the mesenteric glands.
3. Tubercular caseous nodules in the head of the pancreas.
4. Commencement of stricture of the small intestine (ileum,—about twenty inches above the valve). The figure points to a roll of thickened mucous membrane. Beyond that point the mucous membrane is gone, and only the indurated, excoriated, submucous tissue is left, marked by numerous shallow longitudinal fissures.
5. Thickened submucous coat.
6. Greatly hypertrophied muscular coat (circular fibres).
7. Peritoneal coat, and longitudinal fibres of muscular coat.
8. Mass of adipose tissue indurated by a chronic inflammatory process.
9. Deep ulcer leading into the submucous tissue.

was ½—¾ cm. The stricture commenced 9 cm. (three and a half inches) from the sphincter ani. The length of the stricture was 5 cm. (two inches). The opening where the bougie had perforated the wall was 3½ cm. (one and three eighths of an inch) from the lower end of the stricture. That is to say, it was about two thirds through and at a point where the lumen of the stricture changed its direction rather abruptly. The whole of the tissues were very hard. The mucous membrane was ulcerated and in many places gone, but there was no *deep* ulceration. The mesenteric glands were greatly enlarged, many reaching 2½ cm. in diameter. There was a second stricture in the small intestine about the junction of the jejunum and ileum. Length of the stricture 2½ cm. (about one inch). It was very tight and admitted only a No. 12 catheter. On the mesenteric side there was a very deep excavated ulcer 1½ cm. in length, leading into a mass of indurated mesentery, and so perforation was avoided. There was no ulceration of the intestine except at the strictures. This stricture was quite evidently produced by the gradual involvement of the intestine *from the outside* in the tubercular mass in the mesentery. There was a *third stricture* in the ileum, about a foot above the ileo-cæcal valve. It was tight and would only admit a No. 9 catheter. Like the others it was surrounded by a mass of indurated fatty tissue in which several lymphatic glands were embedded. The length of the stricture was 8 cm. (three and a half inches); greatest width when laid open 3½ cm.; greatest thickness of intestinal wall 1½ cm.

The mucous membrane was irregularly destroyed over the greater part of the stricture. The thickening was very dense and hard, and affected chiefly the muscular and submucous coats. There was a deep ulcer leading into the submucous tissue. The mass in the mesentery, which formed part of the growth surrounding the stricture, was 4½ cm. in thickness.

The pancreas had two separate nodules in its head, one 1½ and the other 1 cm. in diameter.

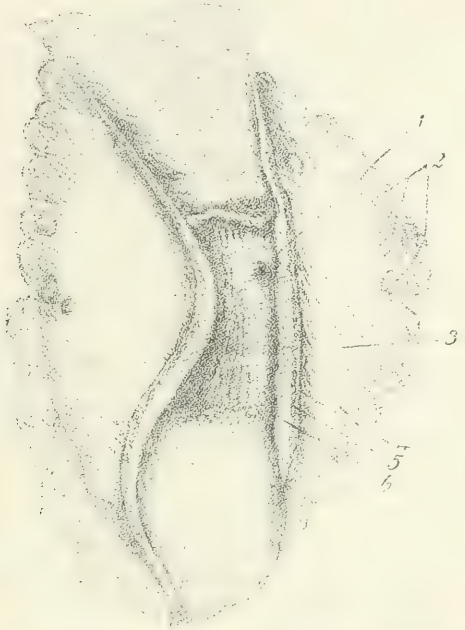
The liver weighed 36½ oz., and had a remarkably elongated, thin, narrow, left lobe. There were no secondary growths and it was not fatty.

The spleen weighed 5¼ oz. and contained two small nodules the size of a pea and a No. 1 shot respectively.

The kidneys weighed 6¾ oz. together, no deposits, normal in appearance, except for the somewhat dilated pelves.

The heart was small but healthy, and weighed 6½ oz.
The brain showed no deposits.
Both lungs were almost universally adherent and were removed

Woodcut 5.



Rectum with stricture, embedded in a mass of indurated adipose tissue, laid open by a longitudinal incision. From a sketch by Dr. Handford. Half natural size.

1. Perforation made by bougie and passing through into peritoneum.
2. Enlarged tubercular lymphatic glands cut across.
3. Mass of indurated adipose tissue.
4. Peritoneal coat.
5. Muscular coat.
6. Greatly thickened submucous coat.

The stricture commences with a roll of mucous membrane as in Woodcut 4. Below that the mucous membrane is gone for the whole length of the stricture, and only the excoriated submucous tissue is left with numerous shallow longitudinal fissures.

with some difficulty. There was a very extensive caseous tubercular infiltration sharply limited to the upper lobes on both sides.

The case was diagnosed at first as tubercular ulceration of the intestine, with possibly tubercular peritonitis of a very chronic form. But when the altered shape of the fæces was noticed, and the very tight rectal stricture was found, carcinoma was suggested, the only points against it being the absence of ulceration of the stricture and the presence of tubercular consolidation of the apices.

A careful microscopic examination was made of the lung, the walls of two of the strictures, the nodules in the head of the pancreas, and of the lumbar and mesenteric glands. No tubercle bacilli were found, but no other conclusion could be arrived at than that the growths were tubercular. Numerous giant-cell systems were found and there was abundant caseation.

In the strictures the thickening appeared to be confined to the submucous and muscular coats, both of which were much thickened; and the muscular coat was freely interspersed with caseous nodules. *April 3rd*, 1888.

P.S.—The specimens are in the Museum of the Royal College of Surgeons, and Mr. Eve, the Pathological Curator, informs me that he has no doubt they are tubercular.

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16. *Three cases of inflammatory changes in the intestinal and mesenteric lymph-glands in diphtheria.*

By Walter S. Colman, M.B.

[With Plate VIII.]

Martha P—, aged 5 years. Admitted December 2nd, 1887, with symptoms of laryngeal obstruction, &c. She had been ailing for three days, and dyspnœa had been present for thirty-six hours. Patches seen on tonsils. There was a trace of albumin in the urine. Tracheotomy was performed, and a tube of membrane removed from the trachea. The obstruction to respiration recurred, and she died cyanosed thirty-six hours after the operation, or five days from commencement of illness. She passed only two semi-solid motions, which were light yellow in colour, "more like jaundice than typhoid." Temperature never above 101°.

Post-mortem thirty-six hours after death.—Posterior surface of soft palate, both tonsils, and whole of larynx uniformly covered by an unusually hard layer of membrane. Below tracheotomy wound, membrane occurred only in scattered patches. Bronchi contained similar patches. There were several areas of consolidation at the bases, shown microscopically to be broncho-pneumonia.

Glands of neck and anterior mediastinum enlarged, matted in œdematous connective tissue, and very hyperæmic.

Kidneys showed, microscopically, early acute nephritis.

Spleen unusually small and very pale.

Liver in condition of cloudy swelling.

Œsophagus quite free from membrane.

Stomach and duodenum healthy.

Jejunum and ileum: Mucous membrane not hyperæmic, but all the Peyer's patches were strikingly prominent, and surrounded by a halo of congestion. They were swollen and extremely hyperæmic, and the vessels leading to them were much engorged. They were more circular than usual. Each was bounded by a continuous raised rim one eighth of an inch in height. The patch was not uniformly raised, but projecting from the floor were several intensely congested convoluted ridges, which formed a coarse irregular reticulation. Both the ridges and the floor were covered by smooth mucous membrane. There was no diphtheritic deposit, nor any ulceration. The colon was normal. *Microscopically*, there was extreme swelling of the mucous and submucous coats, the fibres of the muscularis mucosæ being broken up. The glands of the mucous membrane were also displaced and separated by the abundant small-celled exudation. The small vessels of the mucous and submucous coats were much distended. The follicles in all the sections were quite distinct. The cells in the centre of each were rather swollen and ill defined, and took on staining reaction very slowly. The muscular coats appeared perfectly healthy. Staining for micro-organisms yielded only negative results.

Mesenteric glands enlarged to the size of a pea, red and soft. *Microscopically*, there was some proliferation of cells in the follicles. The blood-vessels were much dilated. There was no hyaline change, but many of them contained a very large number of leucocytes (sections shown). The adenoid reticulum was unusually distinct, and there was swelling and proliferation of the large endothelial cells.

Case 2.—Mabel G—, aged 13 months. Admitted suffering from extreme laryngeal obstruction. One brother died of "croup." Patient said to have been in perfect health forty-eight hours before admission. Tracheotomy was performed, but child died asphyxiated three days from supposed commencement of illness. Temperature never above 101°. She passed two semi-solid, bright lemon-coloured motions, containing a large quantity of mucus.

Post-mortem four hours after death.—There was a large patch of membrane on posterior surface of right tonsil. Epiglottis thick. Laryngeal surface firmly covered by membrane, which was continued over whole of larynx. There was a thin, more recent, and very adherent layer all down the trachea, becoming thicker and more œdematous in the larger bronchi. Patches of collapse at both bases, and at the left base there was a good deal of broncho-pneumonia. Comparatively little enlargement of cervical and mediastinal glands.

Heart normal.

Spleen not enlarged.

Kidneys swollen and hyperæmic.

Intestines: Malposition of the cæcum which was at the level of the umbilicus, the ascending colon being only an inch and a half long. The descending colon was normal in position, relations and length, but the sigmoid was unusually long, and had a very long meso-sigmoid, so that its convexity occupied the *right* iliac fossa.

On opening the small intestine exactly similar hyperæmic swelling of Peyer's patches was found, the mucous membrane itself being quite pale. They were more oval, and in this case also confined to the small intestine. There was no membrane, and no ulceration. The solitary glands were only slightly enlarged. *Microscopically,* the appearances were the same as in Case 1.

The mesenteric glands were very much enlarged, some being the size of a Barcelona nut. *Microscopically,* each follicle of the gland was much enlarged, and there was also extensive cellular infiltration around each. At the periphery of each follicle there was also abundant proliferation, and at the centre of each (as in the centre of each follicle of the Peyer's patches) was a mass of swollen ill-defined cells, some of whose nuclei were proliferating, others clear and swollen, and others broken up and granular. Unlike the rest of the cells in the follicle, they were stained very slightly by logwood.

DESCRIPTION OF PLATE VIII.

To illustrate Mr. W. S. Colman's paper on Intestinal and Mesenteric Lesions in Diphtheria. (Page 122.)

The drawings show extensive acute inflammatory and ulcerative lesions of Peyer's patches. From Case 2, Mabel G—, æt. 13 months.

Case 3 (shown as a recent card specimen, March 6th, 1888).—Thomas S—, aged 3¼. Admitted March 2nd, 1888, suffering from laryngeal obstruction. On February 29th was playing about in usual health. When he woke on March 1st it was noticed that he had a "croupy cough." There was an extensive patch of membrane on the uvula, extending for half an inch on the roof of the mouth. Tracheotomy was performed at 5 p.m., but the child died next day, sixty hours after apparent commencement of the disease. During the operation the child passed a fluid motion of a dark slate colour. He had been taking iron. After this bowels were moved at short intervals until death. Temperature reached 103° a few hours before death.

Post-mortem forty hours after death.—On removing tongue and thoracic organs, membrane was found covering the uvula, tonsils, and posterior pillars of the fauces, both surfaces of the epiglottis, and the whole of the larynx. Below tracheotomy wound, softening of the membrane had occurred, and it was greenish and semi-fluid. The same substance was found extending down the bronchi as far as the base of the right side. Not so far on the *left*.

Lungs rather voluminous and much engorged. Numerous extensive patches of collapse chiefly along anterior border of right lung. Bronchial glands enlarged and very deeply congested.

Kidneys congested and swollen, lobulations ususually persistent.

Spleen congested, not enlarged.

Liver normal.

Stomach and duodenum quite healthy.

Jejunum: There were two intussusceptions in the middle of the jejunum, each about two and a half inches in length, the upper end being invaginated. Each was at the site of a Peyer's patch. On opening jejunum and ileum the following conditions were noted:—There was general redness of the mucous membrane. In the jejunum five feet from the pyloric valve were several brightly injected solitary glands projecting in the valvulæ conniventes.

Below this all the Peyer's patches were found acutely inflamed. There was surrounding congestion, and the patch was raised in an uneven reticulated manner as described in Case 1.

The inflamed patches were confined to the small intestine. The colon was quite healthy.

The three cases just described were three out of four consecutive

cases. The fourth was one which was under observation for over a week with severe and extensive diphtheria of the skin, and secondarily of the larynx, and in this case the intestine and mesenteric glands appeared perfectly healthy.

The association of this condition of Peyer's patches with certain cases of diphtheria is not a matter of recent observation, although very little is to be found on the subject in text-books. Greenfield ('St. Thomas's Hospital Reports,' 1875) gives a careful description of the condition of the intestines in several cases of apparently uncomplicated diphtheria. In all his cases the mucous membrane of the intestine was pale, while the lymphoid structures were intensely injected. All the patches were affected, and the mesenteric glands were acutely swollen.

Recently ('Lancet,' March, 1888) Oertel has described microscopical changes in the intestines and mesenteric glands precisely similar to those in these cases.

Dr. Goodhart ('Diseases of Children') mentions it in passing as one of the rare accompaniments in diphtheria.

In addition to typhoid and diphtheria, scarlatina has been known to give rise to a similar condition of Peyer's patches. Dr. John Harley ('Med.-Chir. Trans.,' 1873) records twenty-five cases, and Dr. Greenfield ('St. Thomas's Reports,' 1875) two cases of apparently uncomplicated scarlatina where this acute swelling of Peyer's patches was observed *post mortem*. *April 3rd*, 1888.

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17. *Ulcerative colitis.*

By W. Hale White, M.D.

Frederick C—, aged 32, was admitted into Guy's Hospital February 10th, 1887, for pain in the abdomen and back together with melæna. The family previous histories were unimportant. Three weeks before admission, without any apparent reason, he had an attack of diarrhœa and vomiting with abdominal pain. A week ago he had to take to his bed, and about the same time vomiting set in. His appetite had gone and he had lost flesh

considerably. On admission, nothing could be detected wrong with the patient by physical examination; there was never excessive tympanites. The bowels were open about ten times a day. The motions were dark red and consisted of fluid containing a little fæces with several small blood-clots, and often some yellowish-looking bodies that appeared to be small sloughs; there was some mucus at times, but the motions could never be called dysenteric. The temperature was about 100° or 101° in the evening. The vomiting, diarrhœa, and wasting continued till the patient sank in about six weeks. At the autopsy it was found that there was ulceration of the whole of the large intestine, beginning at the cæcum and extending to within four inches of the end of the rectum. The ulceration was universal, leaving, however, irregularly shaped islets of swollen, congested mucous membrane. The edges of the ulcers were sharply defined. The floor consisted of exposed transverse muscular fibre. There was no attempt at repair, no perforation, peritonitis or tubercle. The ulceration was least marked in the cæcum and rectum.

This specimen is not a very rare one. We get one or two like it every year among the autopsies at Guy's; yet, considering how very little we know about the condition, I thought it might be worth while to bring it before the members of the Society in the hope that they might be able to throw some light upon it. Dr. Pitt, in the 'Pathological Society's Transactions' for 1885, described a specimen under the title of colitis polyposa which I well remember and which was in many points like this; the ulceration was in his case so deep that the swollen islets of mucous membrane left behind looked like polypi, and hence the name given to the disease. It will be observed that his case was due to dysentery. The causation of my case was very obscure, and we have occasionally cases of ulcerative colitis in which no cause whatever can be made out and which, therefore, must be looked upon as distinct from Dr. Pitt's case.

The College of Surgeons Museum contains no specimen like the one I show this evening. The various books, also, do not help us much with regard to these cases, Leube, in von Ziemssen's 'Cyclopædia,' and Rokitansky, in his 'Pathological Anatomy,' both allude to the condition, and would consider the process might begin in either follicular ulcers or catarrhal ulcers, both of these lesions of the large intestine about which very little seems

to be known as regards cause and symptoms. Nothnagel ('Beiträge zur Physiologie und Pathologie des Darmes,' 1884) does not even allude to the condition.

I am at present collecting all the cases I can find. It would seem to be a process the cause of which is quite unknown, which cannot always be predicted during life, which is incapable of repair, and which rarely leads to perforation. *December 6th*, 1887.

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18. *A case of lymphadenoma in which the intestinal glands were extensively affected.*

By W. B. Hadden, M.D.

I have brought this case before the Society because it exhibits an overgrowth of the lymphatic tissue of the intestines to a very unusual degree.

The patient was a man, aged 54, who was under the care of Dr. Bristowe at St. Thomas's Hospital for some months during 1886 and 1887.

It is unnecessary to give more than a brief account of his symptoms during life. He had been losing weight rapidly for at least a year before admission. On examination, he was found to have a hard, movable tumour in the abdomen below the umbilicus and a little to the right of the middle line. There were the signs of effusion into each pleural cavity and into the abdomen. The lower end of the spleen was felt about the level of the umbilicus. Some of the superficial lymphatic glands were enlarged, but not to any great extent. The lower limbs and the scrotum were œdematous. It was necessary on two occasions to tap the abdomen. The fluid withdrawn was milky but blood-stained; its sp. gr. was 1025, and it contained much albumen. Microscopically, there were leucocytes and red blood-corpuscles, a few large granular rounded cells with large spherical nuclei, and much finely granular material, to which the milky appearance was probably due. On standing the ascitic fluid separated into an upper cream-coloured opaque layer, and a substratum of scarlet clot. Towards the end of life

a quantity of pus-like fluid accumulated in the anterior chamber of the right eye, and bedsores formed.

The temperature was usually normal or subnormal; it was often 95°, and never rose above 100·8°. The bowels were irregular, sometimes open once daily, sometimes not at all, occasionally relieved four, five, or six times in the twenty-four hours. The character of the stools was not noted.

At the *post-mortem* examination the right pleura was found to contain five pints of thin, pus-like fluid and the left pleura a pint and a half. There was no pleurisy. The pericardium contained a little serous fluid. There were signs of general peritonitis, the adhesions being evidently of some standing. Several pints of fluid similar to that seen in the pleural cavities were found in the peritoneal cavity. A large tumour, weighing nearly 14 lbs., was found in the abdomen, involving the mesenteric and retro-peritoneal glands, but the former to a much greater extent than the latter. On section, the mass was pale, rather firm, and studded sparsely with recent hæmorrhages. The iliac, inguinal, cervical and axillary glands were also affected. The urachus was thickened, dilated, and full of what appeared to be inspissated pus. On the surface of the right lung there were some granulations which closely resembled miliary tubercles, and on the left lower lobe there were some yellowish-white areas of new growth. In the liver there was a solitary nodule of growth. The spleen was large, measuring ten inches in length and five inches in breadth. On section, it was dull red, studded with numerous small white nodules, apparently diseased Malpighian bodies, and contained one large old infarct. The kidneys were slightly granular but exhibited no sign of new growth. The pharynx, larynx, and œsophagus were normal. Near the pyloric orifice of the stomach there was one small, raised nodule of growth, about one third of an inch in diameter, apparently arising in the submucosa. All the intestinal glands, solitary and agminated, from the duodenum to the rectum, were much enlarged, elevated, and firm; the enlargement was very marked in Peyer's patches, and in the ileum near the valve the masses were enormous. The growth was strictly limited to the glands, and there was no ulceration. On microscopical examination, the glands of the small intestine showed abundant small round-cells, evidently a hyperplasia of the normal lymphatic tissue. The round-celled growth was localised to the glands, and there was no sign of invasion to neighbouring parts.

Hyperplasia of the lymphatic tissue may of course occur anywhere in lymphadenoma, but it is curious that the intestines are comparatively rarely affected. I myself have seen no other instance in which the intestines were affected at all, and from inquiry into the matter I have come to the conclusion that the case is very unusual in this respect. It is worth noting that the disease in the intestine was strictly limited to the glands, thus differing very materially from a case reported by Dr. Murchison ('Trans. Path. Soc.,' vol. xx, p. 192). In this instance there was enormous thickening of the coats of the duodenum and upper part of the jejunum, apparently due to deposit in the submucous and subserous tissue. Throughout the small and large intestine there were isolated plates of new growth in the walls, causing corresponding elevations of the mucous membrane, but Peyer's patches and the solitary glands were unaffected.

Dr. Sidney Coupland has reported in our 'Transactions' (vol. xxix, p. 363) a case of lymphadenoma which resembles very closely the one I describe. There was uniform hyperplasia of the submucous lymphatic tissue of the intestines, affecting the solitary and agminated glands to an extreme degree, though the growth was by no means limited to the glands but infiltrated the walls of the intestine to a very considerable extent. The intestinal affection was, in Dr. Coupland's opinion, the primary condition. In the case I have just described there was no reason to suspect that this was so.

I should like to direct attention to the nature of the effusion into the serous cavities in my case. The fluid resembled that seen in chylous ascites, and it is quite possible that it was really of the same nature, its miliary colour being due to the mixture of chyle with the ordinary serous effusion. *October* 18*th*, 1887.

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19. *Large rectal polypus.*

By A. Marmaduke Sheild, M.B., B.S.

The specimen was removed by means of the ligature from a woman 42 years of age. For eight years she had suffered from symptoms indicative of the disease. Hæmorrhage

was constant, so that a condition of anæmia was produced, and sometimes profuse bleedings occurred reducing the patient to great prostration. Abundance of offensive and tenacious mucus issued from the anus; the presence of the growth impeded defæcation, and it afterwards protruded from the anus. On several occasions it had become strangulated, causing much pain and difficulty of reduction.

In the recent state the tumour was the size of a small orange, and attached by a thick but soft pedicle of about one inch in length to the mucous membrane of the rectum, about one and a half inches from the anus, on the dorsal aspect of the gut. It was dark in colour, lobulated and foliaceous on the surface, soft and lacerable in consistence, resembling in appearance a portion of pancreas or salivary gland. One of the club-shaped protuberances was examined microscopically, and found to consist of glandular and fibrous tissue, with a covering of columnar epithelium.

Although from time to time specimens of rectal polypi are brought before us here, or in various publications, yet the present specimen is sufficiently interesting, from its size and appearance, to warrant its production and exhibition. From its slow growth, the absence of implication of the mucous membrane at its pedicular attachment, and its minute structure, we are justified in placing it under the category of innocent tumours. The lobulated surface and foliaceous appearance remind us of villous growths of the bladder.

October 18*th*, 1887.

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20. *Intestinal concretions.* (*Card specimen.*)

By W. B. Hadden, M.D.

The specimen shows nineteen distinct calculi. They vary in bulk from the size of a large cherry to that of an orange pip. They differ much in shape, some being pyramidal, some roughly oval or square, and some irregular. The surface is dull white or whitish brown, or of a pale orange tint; some are smooth and some roughened. Several of the calculi are grooved. Two have been bisected,

and in these a large brownish nucleus is seen surrounded by white material.

The patient was a little girl, aged 7, who had suffered for several months from chronic diarrhœa dependent on intestinal catarrh. She was treated during most of this time with chalk in various forms and bismuth.

At the *post-mortem* examination the concretions just described were found in the large intestine, chiefly in the transverse colon. The mucous membrane had been extensively ulcerated by the calculi, and there were numerous pouches. The chemical composition of the concretions was as follows:—Tricalcium phosph. 67·65; Calc. carb. 1·22; nitrogenous matter 23·47; moisture 7·41.

April 3rd, 1888.

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21. *Atrophic cirrhosis of the liver showing irregular nodular prominences due to fatty degeneration.* (*Card specimen.*)

By Leopold Hudson.

The liver was obtained from a woman, aged 58, who had been under Dr. Cayley's care in the Middlesex Hospital. It weighed 47 oz., its capsule was much thickened and its edges rounded. It presented a typically hobnailed surface and was much indurated except at two places where there projected forwards large nodular prominences or bosses which had a smooth surface and soft consistence. Of these the larger, of hemispherical shape, projected from the upper surface of the right lobe, half an inch from the falciform ligament, and one inch from the anterior border; it measured three inches from before backwards, and four transversely. The smaller boss was situated on the upper surface of the left lobe three quarters of an inch from the falciform ligament, and one inch from the anterior margin; it measured two inches in each direction. Section of these nodular projections showed that they consisted of areas of liver-tissue which had undergone fatty degeneration, but had escaped altogether the cirrhotic process which affected the rest of the organ, the line of demarcation between the interlobular fibroid growth and the simple fatty degeneration being plainly

visible to the naked eye and afterwards confirmed by microscopic investigation.

It is well known that the invasion of a tissue by one pathological process frequently protects it from other morbid incursions, and it is suggested that here a preliminary localised fatty degeneration rendered these areas of hepatic tissue capable of resisting the subsequently developed cirrhosis. *December 20th*, 1887.

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22. *Liver, portion of peritoneum, and spleen, from a case of chronic peritonitis with ascites.* (*Card specimen.*)

By George F. Crooke, M.D.

The above organs were removed from a case of chronic peritonitis of two years' standing according to the history obtainable.

The patient, a boy aged 14, was admitted twice into the General Hospital suffering from ascites. The first occasion was on July 12th, 1886, when the abdomen measured twenty-nine inches in circumference at the umbilicus. After a stay in the hospital of two months he was discharged considerably relieved. The diagnosis was chronic peritonitis with ascites. Eighteen months later, on February 7th of this year, he was readmitted with his symptoms much aggravated. The abdomen now measured thirty-eight inches in circumference at the umbilicus, was exceedingly tense, and dull all over. Two days after admission he was tapped, and 465 oz. of an orange-coloured serum was removed. About six days after the operation symptoms of acute peritonitis supervened, and he died on February 20th, thirteen days after admission. At no time was there any enlargement of the liver to be made out; beyond reduplication of the second sound and slight accentuation of the pulmonary second sound there were no symptoms or signs of importance referable to the heart. He had a cough but no expectoration, and there were moist râles heard at the bases. The urine contained a trace of albumen, abundant lithates, but no casts.

Necropsy eighteen hours after death.—Lungs: lower lobes collapsed and carnified; diffuse bronchitis; no pleural effusion or adhesions.

Heart: Adherent pericardium with extreme fibroid induration and calcification of the inflammatory product, the ventricles being encased, as it were, in a cartilaginous and calcified covering. General chronic, as well as acute, peritonitis; seventeen pints of yellow serous fluid containing flakes of fibrinous and purulent lymph removed from the peritoneal cavity. Small intestines matted together, the hepatic flexure of colon firmly adherent to the under surface of the liver; the transverse and splenic flexures of the colon were adherent to the omentum and abdominal wall by semi-organised lymph, the meshes of which were much infiltrated by sero-purulent fluid, forming in the left hypochondrium almost a localised abscess.

Liver weighed only 30 oz. General perihepatitis, the thickened capsule being peculiarly honeycombed and reticulated. The calibre of the portal vein and hepatic ducts much diminished by the thickening and contraction of Glisson's capsule. Liver-substance firm, tough, light brown in colour, with a diffuse ochry mottling, diffuse fibroid change following the distribution of Glisson's capsule throughout the organ. Sections from the peripheral part, including the thickened capsule, and also from the central part of the liver, are shown under the microscope.

Spleen: Similar capsulitis with induration of the organ.

Remarks.—So universal an affection of the serous capsule and also Glisson's capsule at its entrance into the liver at the portal fissure is by no means common in perihepatitis. It has produced unusual shrinking of the organ itself. Microscopically there is great increase of the fibrous connective tissue following the distribution of Glisson's capsule in the liver, and simulating a multilobar cirrhosis. The extension of the fibrous bands into the superficial part of the liver from the capsule has resulted in atrophy of many lobules. The liver-cells in the interior of the organ appear in good condition, and their nuclei stain well. Perihepatitis of the liver is mostly localised, and Hilton Fagge says it is rarely accompanied with a widely distributed cirrhosis.

May 1st, 1888.

Fig 1

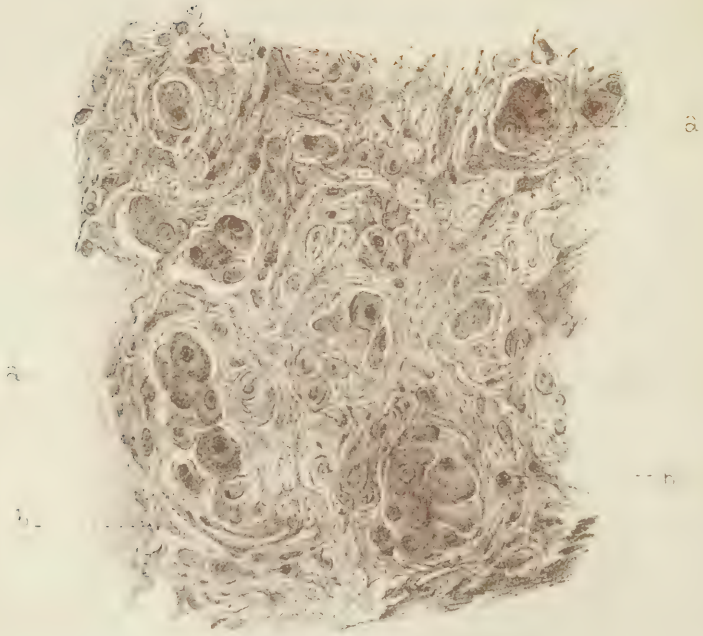

Fig 2

DESCRIPTION OF PLATE IX.

To illustrate Dr. Penrose's specimen of Pericellular Cirrhosis of the Liver. (Page 135.)

From drawings by A. Stone.

FIG. 1.—Section through the right lobe of the liver.

a. Remains of liver-substance but little altered.

b. Minute white specks entirely consisting of the interstitial growth (miliary gummata).

FIG. 2.—Section through yellowest portion near the edge of the liver.

a. Hepatic cells.

b. Cells of interstitial fibrous overgrowth.

23. *Pericellular cirrhosis of liver in a case of congenital syphilis.* (*Card specimen.*)

By F. G. Penrose, M.D.

[With Plate IX.]

Naked-eye appearances.—The liver occupied the upper half of the abdominal cavity, and the edge of the right lobe extended into the right iliac fossa. There was no perihepatitis. Weight 20 oz.

Surface smooth, of a brilliant yellow colour, spotted irregularly with fine white dots all over the anterior surface of both right and left lobes. At the thickest part of the right lobe the surface is mottled from the presence of liver-substance of almost normal appearance. The edge is hard and thickened.

On section, the tissue is very firm, the greater portion of a brilliant yellow, speckled over with fine white dots similar to those seen on the surface, and no distinction into lobules can be made out even with a hand lens. In the centre of the thickest part of the right lobe a portion of liver-substance still remains of almost natural appearance and consistence. No evidence of albuminoid change.

Microscopical appearances.—There is general diffuse fibrous overgrowth separating the liver parenchyma cells into small groups of one, two, or more. This fibrous growth does not appear to have any definite relation to the portal system. The white specks mentioned above are found to consist of masses of this fibrous tissue, in which there are no remaining liver-cells, and which may be considered as miliary gummata. This interstitial growth is only sparsely nucleated, and only very slightly vascular.

Subjoined is a history of the case.

G. L—, aged 10 weeks, came under the care of Dr. Cheadle at the Hospital for Sick Children, Great Ormond Street, on February 5th, 1887.

Past history.—The child was born at full term, fed entirely on cow's milk, and had been wasting rapidly during the last fortnight. Five days before admission mother noticed a rash about nates, and for three days child had been suffering from constant diarrhœa.

No vomiting. No history of syphilis could be obtained from the mother.

State on admission.—Weight 8½ lbs.; emaciated. Skin rough, sallow, of a muddy *café au lait* colour. Conjunctivæ distinctly yellow. No œdema. On buttocks and about the scrotum the skin is excoriated, and there are recent fissures about the anus; no sign of any rash elsewhere. The child is drowsy, takes no notice, seems unable to cry. No snuffling. Lips bluish; tongue clean; buccal mucous membrane of a yellowish tinge. Abdomen distended; resonant in the left flank. The liver is enormous; the edge reaches down into the right iliac fossa and crosses the abdomen below the level of the umbilicus, and is lost behind left margin of thorax behind the eighth costal cartilage. The spleen is much enlarged; the edge is felt two and a half inches below the margin of the thorax.

The child gradually sank, and died four days after admission. Whilst in hospital the physical signs remained unchanged. Diarrhœa continued; averaged seven motions daily. Stools liquid, of a pale yellow colour. Temperature was raised, and varied from 99·4° to 102° F.

Post-mortem.—Some fat in the subcutaneous tissues; skin abraded and wrinkled about anus.

Liver: See description.

Spleen enlarged; weighs 3 oz.; very dark in colour, very firm in texture. Under the microscope nothing abnormal could be seen.

Kidneys natural size; very tough; of a pale yellow colour. Capsule strips away readily enough. On section, the distinction between cortex and medulla is difficult to see by the naked eye.

Suprarenals large; of a dull red grey colour, quite unlike ordinary suprarenal colour.

Stomach empty of food; contains a thick, tenacious, dark brown mucus. The mucous membrane is perfectly white. Intestines very pale. Mesenteric glands enlarged, and spotted over with little yellow dots.

No pleurisy. Lungs: Lower lobes collapsed; no consolidation. Larynx and trachea very pale; contain a yellowish red mucus.

No pericarditis. Natural quantity of normal-coloured pericardial fluid.

Heart: No petechiæ on the surface, which is tinged pale yellow. Right auricle distended by a pale firm yellowish clot. Right ven-

tricle dilated by a large pale yellow clot. Wall yellow, thin, and very tough. Tricuspid and pulmonary valves healthy. Left auricle contains a small, pale, firm clot. Left ventricle dilated by yellow clot. Ventricular wall thin, yellow, tough. Mitral and aortic valves healthy. The internal surface of the aorta is of a bright golden yellow colour.

Brain pale and firm. Fundus of eyes perfectly healthy.

Remarks.—Although the condition of the liver above described is well known, and is described in most of the text-books on pathology, and in the 'Transactions' of the Society are papers by the following authors referring to the same or closely allied conditions: Dr. Samuel Wilks, vol. xvii, p. 167; Dr. Hermann Weber, vol. xviii, p. 133; Mr. Symonds, vol. xxxvii, p. 124; and in vol. xxvii, p. 303 Dr. Coupland describes a large gumma of a brilliant orange-yellow colour, yet I have thought it worth while to bring this specimen before the Society on account of the extreme degree to which the interstitial changes have gone, and the remarkable appearances of the various organs in a fresh state.

December 20th, 1887.

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24. *A case of primary carcinoma of the liver.*

By George F. Crooke, M.D.

[With Plate X.]

For the privilege of bringing before the notice of the Society the following case of primary growth in the liver, as well as for the card specimen shown, I am indebted to the courtesy of Sir W. Foster and Dr. Simon, under whose care the cases came.

The subject of the primary growth in the liver was a man aged 65, a painter by trade, and there are one or two facts about his family and personal history worthy of mention. His father died from a tumour on the front of the chest, caused, it was thought, by a kick from a horse. One sister died of phthisis.

Forty-two years ago the patient violently sprained his back, and was laid up three to four months in bed with paraplegia. Ever since then he has experienced lumbar pain on any effort at exertion.

Eight years ago he had right-sided pleurisy. In July of last year he was an in-patient in the hospital on the surgical side with an ulcerated leg, and was discharged in September.

For some weeks before admission he was under Dr. Simon's observation as an out-patient, and an attack of bronchitis, with chills and severe pain over the region of the liver, compelled him to seek admission on January 20th of this year. At this time enlargement of the liver was observed, and Dr. Simon sent him in for admission on the above-named date.

He was a sallow-complexioned man, but with a decided icteric tinge of the skin and conjunctivæ, but fairly well nourished. The abdomen was greatly enlarged, tense, tympanitic in front but dull in the flanks, and there was some amount of ascites. The liver was uniformly enlarged, its lower edge, hard and sharply defined, could be easily felt two and a half inches below the costal margin. From the ensiform cartilage to the lower border of the liver the measurement was five inches, so that the anterior surface of the organ could easily be palpated over this area. It felt comparatively smooth but very hard, and there was much tenderness on palpation. The patient experienced a constantly dull aching pain over the region of the liver, both in front and behind. He suffered also much from dyspeptic symptoms, anorexia, pain after food, great thirst, with dryness of the mouth and tongue.

His further history shows that his weakness increased; he began to emaciate, his jaundice and ascites became more pronounced, and were accompanied by considerable effusion into the right pleural cavity, from which 40 oz. of bile-stained serous fluid were syphoned off. Exploration of the abdominal cavity with an injection syringe drew off some green-coloured opalescent fluid rich in albumen and chlorides, which under the microscope was found to contain a few red blood-cells, numerous leucocytes, but no hydatid hooklets. Exploration of rectum for primary growth negative, neither were symptoms present pointing to any such growth in any part of the alimentary tract.

The patient had not been a drinker. Sir W. Forster and Dr. Simon diagnosed the case to be one of cancer of the liver. The patient gradually sank from asthenia.

At the necropsy the liver was the only organ affected with growth, and though careful search was made, no other primary source could be found. Its convexity, corresponding to the vault

of the diaphragm, to which it was adherent, reached as high as the level of the third interspace. It is uniformly enlarged, weighing 104 oz.; its transverse diameter measured fourteen inches, its antero-posterior eleven, and its thickness four inches. The general shape is unaltered. Its entire external surface is uneven by the presence of numerous circular nodules of growth projecting beneath the capsule. These nodules in the fresh condition were white and reddish grey in colour, varying in diameter from one tenth to one half of an inch, sometimes quite discrete, sometimes confluent. The smallest are white and pearly looking, and the larger are distinctly umbilicated in the centre.

On section, the growth is more or less diffused in the central part of the right lobe, where hardly any traces of liver substance remain; it is tough and fibrous, grey white in colour, but streaked and mottled with yellow lines, indicating fatty changes. At the extremity of the right lobe, and throughout the whole of the left the discrete nodular infiltration prevails; each larger nodule is surrounded by a somewhat vascular zone shading off into the intervening liver structure, which is of a greenish-yellow colour from bile staining, and no fibrous capsule could be made out.

Very small raised nodules, about the size of a millet seed, were found in the peritoneal lining of the diaphragm.

The gall-bladder is atrophied, empty, and its walls thickened; the cystic duct appears blocked by the pressure on it of a small, hard, calcareous gland, about the size of a small marble. One or two other portal glands were found hard and infiltrated by the growth. Bile-ducts patent, and no growths were found in the portal vein. Seventeen pints of bile-stained fluid were removed from the peritoneal cavity. No secondary growths were found in other organs.

The inferior lobe of the right lung was collapsed, carnified, and airless, and two pints of bile-stained serum were found in the pleural cavity.

No information as to the origin of the growth could be obtained from histological examination of the centre of the right lobe, where the growth was very diffuse and fibrous in character, and hardly a trace of liver structure discernible. The appearances here were islets of the fully developed growth (to be afterwards described, Pl. X, fig. 4), luxuriant and staining well, alternating with similar patches in an advanced stage of atrophy and degeneration.

The relations of the liver-cells to the growth are best studied at the periphery of small discrete nodules, about one tenth of an inch in diameter, and separated by a fair amount of liver substance. The following are the appearances to be observed on examining sections taken from such a part: (1) Groups of liver-cells are met with, distinguished by the extraordinary size of their nuclei, which stain deeply with hæmatoxylin; and one or two of them may be seen to be dividing. (2) Next ensues an active division of the nuclei, in which I have been able very well to follow out several of the stages of karyokinesis (figs. 1 and 2). (3) With this nuclear division, vacuolation and division of the liver-cell itself occurs, and so at the periphery of the smaller nodules we find columns of liver-cells in which these processes are in active progress, and the transformation of the liver-cells into those of the growth is quite striking (figs. 2 and 3).

Lastly, the bridges between contiguous vacules become absorbed, while the nuclei arrange themselves at the periphery; in this way a column of liver-cells becomes converted into a tubule lined with a highly nucleated protoplasmic border, or even filled with proliferating epithelioid cells (fig. 3).

Now, a tubule thus formed sprouts out in all directions, the nuclei all the time going on actively proliferating, until we arrive at the fully-developed growth, as illustrated by fig. 4, drawn from the centre of a small nodule one eighth of an inch in diameter, in which no degenerative changes have as yet occurred. This is seen to be composed of a number of irregularly branching tubules separated by a fibro-nucleated stroma, and either lined or filled by proliferating epithelioid cells (fig. 4).

The growth, which I would term an adenocarcinoma, repeats itself in the infiltrated portal lymphatic gland, and in the little millet-seedlike nodules in the subserous peritoneal lining of the diaphragm.

The other points to be considered are:

(*a*) The stroma: This I believe to be derived from the perilobular and intercellular connective tissue. Although it contains spaces and channels, which no doubt serve to convey nutrient plasma to the growth, red blood-cells are rarely met with in the smallest nodules. Only in the larger ones, involving several lobules, where Glisson's capsule enters considerably into the formation of the stroma, are well-defined blood-vessels present.

DESCRIPTION OF PLATE X.

To illustrate Dr. G. F. Crooke's specimen of Primary Carcinoma of Liver. (Page 137.)

From drawings by himself.

FIG. 1.—Liver-cells with dividing nuclei at the peripheral part of small nodules of growth. Hartnack, No. 9, oc. 3. (600 diam.)

FIG. 2.—To illustrate the changes in the liver-cells at the periphery of the smaller nodules of growth.

a. A few normal liver-cells.

b. Liver-cells with large dividing nuclei. b^1. A nucleus splitting up into numerous divisions.

c. Vacuoles in a column of liver-cells.

d. Intercellular stroma, nucleated.

e. Intercellular capillary.

Hartnack, No. 7, oc. 3. (300 diam.)

FIG. 3.—Columns of liver-cells in progressive stages of transition into the cells of the growth.

a. A few normal liver-cells.

b. Liver-cells with large proliferating nuclei dividing.

l. Leucocytes in a vacuolated space.

e. Intercellular capillary blood-vessels.

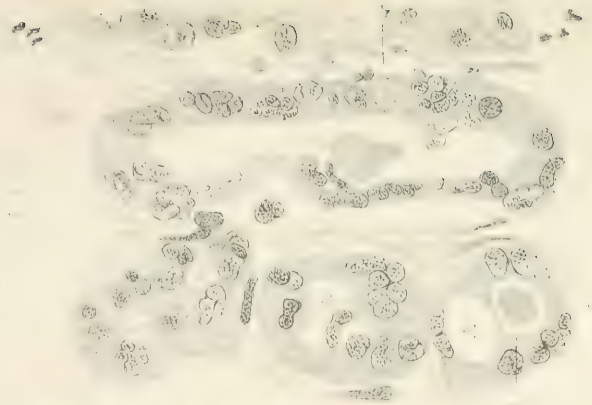
v. Vacuoles.

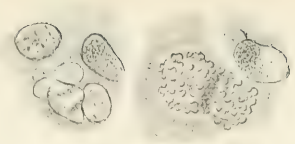
F. Column of liver-cells in which the nuclei are actively proliferating and the protoplasm of the liver-cell becoming at the same time vacuolated (*v*).

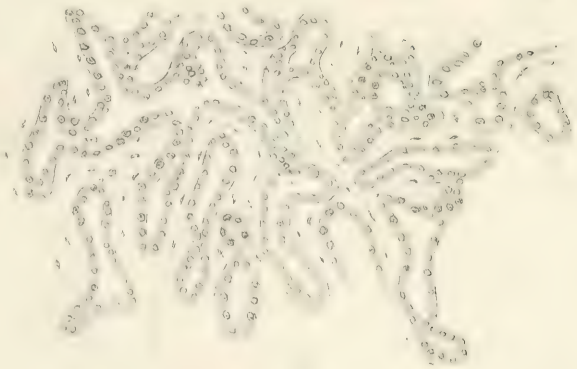
G. A column of liver-cells in which the bridges between the vacuoles have become absorbed. The proliferating nuclei are all arranged peripherally so that the column has become tubulated, *i. e.* resembles a tubule lined with a highly nucleated protoplasmic border.

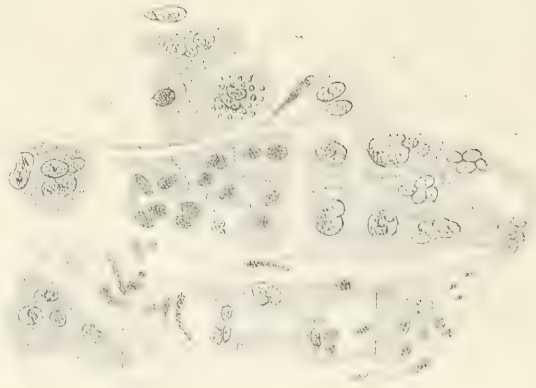
i. Blocks of colloid material resulting probably from degenerated epithelium. (400 diam.)

FIG. 4.—From the centre of a small nodule one eighth of an inch in diameter. It shows the fully-developed growth. Hartnack, obj. 5, oc. 2, tube out. (140 diam.)









(*b*) *Secondary changes in the growth* are increase in the fibrous stroma and fatty degeneration of the cells, the whole breaking down eventually into a structureless débris.

(*c*) *Changes secondary to the presence of the growth.*—The bile-ducts show irritative proliferation of the nuclei of their lining epithelium, simply as a result of local obstruction to the biliary circulation as in biliary cirrhosis. The intercellular biliary canals are here and there seen much dilated and filled with green-coloured bile. Groups of liver-cells are seen to be undergoing pressure atrophy, both from the encroachment of the growth itself, as well as from localised stases of the intercellular blood capillaries.

Records in medical literature of cases of primary growths of the liver are now fairly numerous, and not a few are to be found in the volumes of the Society's 'Transactions.'[1]

Up to the time of Waldeyer's important paper in 'Virchow's Archives '[2] on the Development of Cancer, primary growths of the liver were believed to originate almost exclusively from the bile-ducts.

Since that time another mode of origin has been described by various authorities,[3] viz. from the liver-cells.

Although in this case it is not possible conclusively to determine the origin of the growth in that part of the liver which in all probability was its primary seat, viz. the centre of the right lobe, yet the manner in which the liver-cells everywhere contribute to its formation in its diffusion throughout the organ is certainly remarkable and demonstrated beyond all doubt, and would moreover almost seem an argument in favour of the first beginnings of the growth in this way.

The diffusion of the growth appears to take place by the portal veins as well as by direct contiguity. Many of the veins are seen dilated and filled with the cells of the growth, and thus, as suggested in Prof. Coats' criticism on the subject, some influence is propagated to the liver-cells stimulating them to proliferation.

[1] Greenfield, 1874, vol. xxv; Mahomed, vol. xxviii; Paul, vol. xxxvi; Tivy, vol. xxxv; Pye-Smith, vol. xxxi; G. N. Pitt, vol. xxxviii; Fowler, vol. xxxiii.

[2] "Die Entwickelung der Carcinome," 'Virch. Arch.,' Bd. 51 and 55.

[3] Perls, 'Virch. Archiv,' 56; Dreschfeld, 'Journ. Anat. and Physiol.,' 1880; Fetzner, 'Beiträge zur Histologie des Leberkrebs,' Tubingen, 1868; Harris, "Ueber die Entwickelung des primären Leberkrebs," 'Virch. Arch.,' Bd. 100; with full references to the literature on the subject.

The fully-developed growth is atypical, and bears no resemblance to certain adenomata of the liver described as occurring in cirrhotic livers by Paul[1] and Kierner and Kelsch[2] (see their drawings and papers). It corresponds very closely with that described by Dr. Pitt[3] as a primary scirrhus of a tubular type. *May 1st*, 1888.

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25. *Carcinoma of common bile-duct. Microscopic sections from two cases of primary new growth in the wall of the common bile-duct.*

By Norman Moore, M.D.

Case 1.—From a man, aged 47, a patient under my care in St. Bartholomew's Hospital.

The section is made through the wall of the common duct about half way between its commencement and its terminal orifice. It seems to be developed from the large flattened epithelial cells of the duct, and as it extends outwards into the duct wall the cells appear in the meshes of well-developed but not very dense stroma. In this are included many cross sections of the mucous glands which open into the duct, and in the midst of the new growth are also a few imperfectly developed mucous glands.

Towards the liver the new growth penetrated the hepatic substance at a few isolated points and only for a very short distance. To the naked eye no infiltration of the liver was visible, and there were no secondary deposits in it.

The lumen of the common duct was wider than natural, and the epithelial surface was very faintly bile stained, almost white. The duct contained a good deal of mucus, but a probe was easily passed down it from the papilla. The gall-bladder was greatly distended with bile. Its walls were not thickened. There was no general dilatation of the ducts in the liver, and no general increase of connective tissue.

Deep general jaundice was present, and the body was much

[1] 'Path. Trans.,' vol. xxxvi.

[2] 'Archives de Physiologie,' 1876.

[3] 'Path. Trans.,' vol. xxxviii.

wasted. There was no dropsy. The brain was normal, and there was no new growth in head or neck. There was no new growth in the chest.

The lungs were firmly adherent at their apices and both apices contained tubercle.

The heart was normal, and the aorta and large blood-vessels were not anywhere calcified.

The glands of the hilum of the liver contained no new growth, and none were found in any other abdominal lymphatics.

The whole alimentary tract was free from new growth.

The spleen was somewhat enlarged and soft, weighing 10 oz.

The pancreas was minutely examined, and found to be normal throughout.

The kidneys, suprarenals, ureters, and bladder, and urethra were all normal, and contained no trace of new growth.

The cartilages of the knees and great toe joints on both sides were free from degeneration or deposit.

The patient was a machine fitter. His jaundice began on or about June 12th, 1886. He had never had jaundice or any serious illness before. The jaundice came on after a few days of pain at the epigastrium after taking food, and without any attack of colic. He stated that he had been losing flesh slightly for two years.

He was very thin when admitted to St. Bartholomew's Hospital on July 22nd, and deeply jaundiced. His temperature was normal. The edge of the liver was not to be felt, nor was the spleen enlarged.

On August 1st his jaundice was not diminished, but about August 11th it became a little less intense, and some bile was observed in the stools. On August 18th they were again quite without bile.

On September 10th he was sent to a convalescent hospital. He weighed 9st. 11lb. on July 27th, and 9st. 4lbs. on September 7th.

On October 13th he was readmitted, obviously much thinner, and weighing 8st. 4lbs.

He was still deeply jaundiced. The gall-bladder could be distinctly felt, and slowly increased in size.

He grew steadily weaker, and died November 8th.

Case 2.—From a woman, aged 63, who was under my care in St. Bartholomew's Hospital.

The section is made through a dense whitish mass of new growth

which occluded completely the common duct and the cystic duct. It shows a carcinomatous growth with very dense fibrous stroma, and scattered collections of flattened cells of epithelial type.

The cavity of the duct was discoverable, though its walls were in contact, and the new growth extended from it. The liver was only penetrated in the immediate neighbourhood of the duct.

The gall-bladder itself was occluded, being adherent to the walls of a small hydatid cyst containing creamy, caseous material, but its fundus contained no new growth. There were no secondary deposits in the liver.

The bile-ducts were dilated throughout the liver, and the organ itself was cirrhotic, and of a dark green colour. There were no secondary growths in any part of the body.

The patient was a married woman who had had nine children. She had had pain at intervals in the epigastrium for two years, and after losing flesh for two months became jaundiced, June 15th 1886.

On her admission, on July 6th, she was very deeply jaundiced. The edge of the liver could not be felt. Some weeks later the jaundice was deeper and the gall-bladder (as it afterwards proved to be) was felt as a hard lump at the lower edge of the liver.

She became more and more jaundiced and weaker, and died December 4th, 1886.

These were both cases of primary carcinoma of the common bile-duct. In the man the absence of any history of colic or of any tumour except the distended gall-bladder made the diagnosis difficult during life. At the *post-mortem*, but for the microscopic examination of the duct, the new growth might have been overlooked, and it seems likely that some cases (in which no such examination was made) recorded as examples of chronic catarrh of the bile-duct were really examples of new growth.

In the *post-mortem* room I have never seen an example of chronic catarrhal jaundice.

The case of the woman shows the further results of the growth viz. cirrhosis of the liver caused by the consequent dilatation of the ducts, and illustrates the direction in which primary carcinoma of the common bile-ducts spreads viz. directly into the surrounding structures without remoter infiltrations, even the glands of the hilum of the liver not being affected. This form of new growth is rare, and its nearest ally is to be found in the pancreas, where

primary carcinoma also begins, in many cases, in the epithelium of the ducts, and exhibits the same strictly localised character, only spreading directly into contiguous tissues.

In the pancreatic duct, as in the common bile-duct, calculus frequently exists, and may be considered an exciting cause of the new growth.

In both the cases described in this paper calculus was absent. No definite origin can be assigned in the case of the man. In the woman the hydatid ultimately destroyed, and caseating in the gall-bladder may have given the initial irritation which terminated in the carcinoma.

The duration of the growth deserves note; it was clearly slow in both cases.

In the man the symptoms extended over two years and six months, and he died five months after the appearance of jaundice.

The woman had symptoms for the same period, and died at about five months after the new growth was advanced enough to cause jaundice.

The morbid anatomy demonstrates how difficult the diagnosis of such cases must remain. It seems to afford grounds for the opinion that primary carcinoma of the common bile-duct has a duration of about two years and a half, and shows that it may in that time cause death by general exhaustion with or without producing cirrhosis of the liver. *January 17th*, 1888.

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26. *Cancer of common bile-duct.* (*Card specimen.*)

By J. A. Ormerod, M.D.

Just below the junction of the cystic and hepatic duct the common duct was blocked by a mass of soft whitish, sprouting new growth. For a short distance above this the mucous membrane was granular and thickened. The ducts above were enormously distended, and contained—the cystic duct, thin puriform fluid, and a mass of pultaceous débris, the hepatic duct, a soft yellowish mass (of bile-stained mucus ?). The common duct below the growth

was large, but its mucous membrane was natural. Externally the coats of the gall-duct were natural.

Pancreas natural, could be dissected clean off the gall-duct; pancreatic duct normal.

Gall-bladder distended with thin puriform fluid, and contained several gall-stones. No stones in the ducts.

Liver swollen and greenish, ducts dilated; no new growths. Except for a fibroid tumour of uterus there were no other new growths in the body. Bright jaundice; no ascites.

Microscopically, the tumour proved to be encephaloid cancer (Mr. D'Arcy Power).

The patient, a female aged 58, died in St. Bartholomew's Hospital under the care of Sir Dyce Duckworth, on February 14th, 1888; jaundice, preceded by loss of flesh, nausea, and vomiting, had developed gradually in the previous October. No history of biliary colic. She had pericarditis, febrile symptoms and delirium before death. *May* 15*th*, 1888.

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27. *Hydatids of spleen, liver, brain, etc.* (*Card specimen.*)

By Bernard O'Connor, M.D.

Male patient, aged 37. Profuse hæmoptysis (?) three years ago. Moderate drinker. Until about a couple of weeks before admission, on March 13th, 1888, into the North London Consumption Hospital, he had been in the Middlesex Hospital, where he had an attack of jaundice preceded by severe abdominal pain which had lasted a fortnight. No history of any strain. Bad cough. Aphonia. Occasional hæmoptysis, œdema of feet and malaria. Greatly emaciated. Dyspnœa on exertion. Dry tongue. Cachectic appearance. Jaundiced. Abdomen hard and much enlarged by prominent tumour. No fluctuation. Considerable mapping out of superficial abdominal veins limited by costal margins. Died May 5th.

Post-mortem.—Liver: Three large hydatid cysts (one = one and a half pints).

Spleen: One cyst occupying whole extent of organ, which is reduced to a mere, but firm, shell holding about two pints.

Brain: One cyst about the size of a small pea, in the left choroid plexus.

Lungs: Old cavities at apices, semi-solid below.

Jejunum: Ulcerated patches in lower part.

May 15th, 1888.

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28. *A case of carcinoma of the pancreas and semilunar ganglia.*

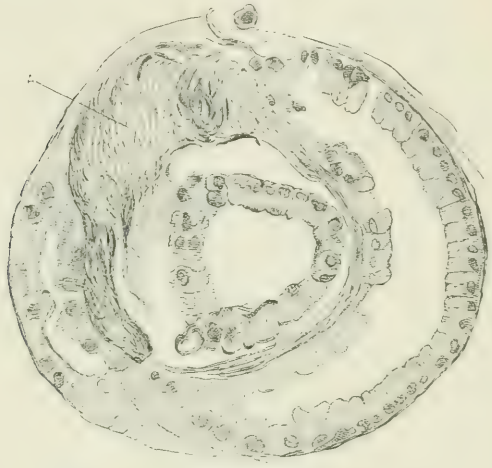
By W. Hale White, M.D.

Ann G—, aged 54, was admitted into Miriam Ward, Guy's Hospital, on June 25th, 1887. Two years before admission she began to complain of vague abdominal pain. Three months ago vomiting set in, and she noticed that she was losing flesh. The only points to be observed on admission were her somewhat wasted condition, which was not, however, more than might be explained by the vomiting, some abdominal pain and tenderness, rather vague in position; and on the flexor surfaces of all four extremities, on the abdomen and in the axillæ, were numerous petechiæ. After a few days' stay in the hospital she gradually sank and died as a result of the continual vomiting. The *post-mortem* examination showed a carcinomatous growth to exist in pancreas extending for its whole width, but leaving the tail and the head free; above it had invaded the connective tissue about the cœliac axis; below there was a fibrous band binding down the small intestine at a spot six inches below the end of the duodenum. The calibre of the gut was here reduced to the diameter of the forefinger. The growth was very hard.

Examination with the microscope showed it to be a typical scirrhus carcinoma. It had slightly invaded both suprarenal capsules and had grown into the semilunar ganglia, the dense fibre tissue of which seemed, however, to present a considerable bar to its progress. The nerve-cells were granular and slightly pigmented but filled their capsules fairly well; their nuclei were not very evident. Some bundles of nerve-fibres were healthy, but at many parts of the section it might be seen that the circular space in the connective tissue of the ganglion in which a bundle of nerve-fibres

lies was occupied partly by degenerated nerve-fibres, and partly by carcinomatous cells. It was clear that the epithelial cells, once having eaten their way into the sheath of the nerve, grew along the nerve inside the sheath, this direction offering less resistance than the dense connective tissue. At some places the epithelial cells might

Woodcut 6.



Transverse section of a bundle of nerve-fibres in the semilunar ganglion. The only unaffected nerve-fibres are at A. They are cut obliquely. The rest of the bundle is occupied by cancer-cells which have grown along it. Some have dropped out.

be seen growing into the sheath of the nerve (Woodcut 6). With the exception of the lesions described the whole of the body was perfectly healthy.

This is a case of very great interest. Primary carcinoma of the pancreas is very rare, and it is to be noted that in this case it was confined to the middle of the gland. But the greatest interest lies in the affection of the semilunar ganglia. Very few, if any, cases of malignant disease affecting these organs are on record, and we learn from this specimen the peculiar mode of growth along the bundles of nerve-fibres. I have examined many hundreds of sympathetic ganglia and have shown elsewhere that the normal condition of the nerve-cell in the human subject is much the same as we observe in this specimen, so that the granular condition here seen must clearly not be ascribed to the invasion by the growth.

Of the symptoms the vomiting may be explained by the obstruction of the intestine. The wasting was not more than the vomiting and the presence of malignant disease would explain. Pain is common to malignant growths in any part of the body, and petechiæ are liable to be present in patients in an underfed condition. It has long been known that growths in the pancreas produce no symptoms recognisable during life, but this case seems to teach us in addition that the semilunar ganglia may be implicated by growth and yet that that no symptoms will be occasioned by which the affection of the ganglia can during life be recognised, although it is possible that in this patient some of the symptoms which can be explained by the contraction of the intestine were really due either to the affection of the pancreas or semilunar ganglia. But I have placed the case on record because it is some evidence that what I have often maintained is true, viz. that, whatever function the sympathetic ganglia may have in the lower animals, they are in us degenerate organs like the coccyx, that are dying out both functionally and structurally, for in this case it is extremely probable that their invasion by malignant disease produced no symptoms. *October* 18*th*, 1887.

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29. *Cyst of the peritoneum.* (*Card specimen.*)

By C. Stonham.

(With Plate XXXVI, fig. 2.)

The specimen was found in a male subject brought into the dissecting room of University College. It was situate close to the right internal abdominal ring, and was hanging down in the pelvic cavity. The attached end of the cyst is in the subperitoneal tissue; it has been opened in order to distend the cavity, but was closed when the specimen was removed. The cyst was partially collapsed and contained blood-stained serum, the staining being probably due to *post-mortem* changes.

The cyst measures six inches and three quarters in length and

about three inches and a half at its broadest part when distended with fluid; close to the peritoneum the neck is about a quarter of an inch broad, but the part situate in the subperitoneal tissue is about half an inch across.

The wall is about the thickness of tissue paper, except that part under the peritoneum, where it is one-twentieth of an inch thick. It is capable of containing about 11 oz. of fluid. The attached end is situate in the subperitoneal tissue, and the cyst projects through a round opening about three sixteenths of an inch in diameter in the peritoneum, being attached to the margins. The cyst wall is continuous with the inner surface of the thickened part in the subperitoneal tissue and not with the outer layers of the peritoneum. The thickening of the neck is due to overgrowth of the connective tissue surrounding it.

Microscopic examination.—A piece of the wall of the cyst examined under a $\frac{1}{6}$th-inch glass and stained with logwood shows a very delicate network of connective tissue, containing in its meshes numerous nucleated cells varying much in shape, but being for the most part rounded or fusiform, some of the latter having a long process connected with one end of the cell. The cells are scattered irregularly, and there is no evidence of an epithelial layer on either surface. A nitrate of silver preparation does not show any epithelium, but the specimen had, before staining, been preserved in spirit.

The wall at the neck of the sac, *i. e.* beneath the peritoneum, is composed of fibrous tissue, surrounded on the outer surface by delicate connective tissue. There is no evidence of epithelium on either surface.

I am indebted to Mr. Raymond Johnson for kindly preparing the microscopic specimens for me.

It is probable that the cyst originated in the subperitoneal tissue, and producing absorption of a small part of the peritoneum overlying it, projected through the membrane into the abdominal cavity. The absence of epithelium would thus be accounted for.

(University College Museum, No. 2225A.) *May 15th*, 1888.

V. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

1. *Hypertrophy of right kidney; congenital (?) atrophy of left.* (*Card specimen.*)

By H. Handford, M.D.

The specimen was taken from a man, aged 47, who died of heart disease and dilatation of the aorta. He presented no symptoms of renal disease during life, and there was no history of antecedent kidney mischief. He passed an average quantity of urine free from albumen.

At the inspection *post mortem* the right kidney was found uniformly enlarged, but preserving a normal contour. The capsule stripped readily, and on section the pyramids and cortex appeared healthy. The weight was $13\frac{1}{4}$ oz., length (after two months in spirit) $15\frac{1}{2}$ cm. (six inches and an eighth). The adrenal was normal in size and appearance.

On the left the adrenal was also of normal size and position. The left kidney was represented by an elongated mass of fibrous tissue, about two inches in length, which contained traces of gland tissue and numerous rounded, hard, yellow nodules, which were encapsuled and consisted of partially calcified, atheromatous material. There was a small renal artery and vein on the left side in the normal position. The ureter, too, was quite normal in appearance and position, and terminated in a saccular pelvis, somewhat smaller than natural.

The atrophy was evidently due to some long antecedent inflammatory disease.

The large size of the hypertrophied kidney is remarkable. It exceeded by *at least* $3\frac{1}{4}$ oz. the average weight of the two normal kidneys in the adult. *April 3rd*, 1888.

2. *A case of congenital hydronephrosis.*

By Dawson Williams, M.D.

George S—, aged 5½ years, was brought to my out-patients at the East London Hospital for Children, Shadwell, on February 23rd, 1886. His mother stated that he had always been a delicate child, and had been suffering from diarrhœa for several months. He had been taken suddenly ill on February 4th, and had been judged to be deaf and blind for a short time. He remained in a "stupefied state" all the next day, and at night had convulsions affecting both sides, and lasting twenty minutes. After an interval of ten minutes he had another convulsive seizure, which did not completely cease for an hour; he was better on the following morning, and the convulsions had never recurred. He had "wet the bed every few minutes" for nine months, but had never complained of any pain or difficulty in micturition. About six months before he came to the hospital it was noticed that the lower part of his belly was swollen and that he was passing an unusually large quantity of water; since that date the swelling of the abdomen had constantly increased, and he suffered much from thirst. There was no history of stone.

He was a pale sickly child, the tongue was furred, he had no appetite, and complained of thirst and headache. In the middle line, immediately above the pubes, was a rounded pyramidal tumour, reaching almost to the umbilicus; the tumour appeared to be the distended bladder. The patient was admitted under the care of Dr. Donkin, to whose kindness I am indebted for permission to peruse the notes taken by Mr. Richards, resident clinical assistant.

There was no phimosis, and a catheter was passed without difficulty, but only a very small amount of urine could be drawn off, and this did not materially affect the size or character of the tumour. He was found to be passing large quantities of pale urine, and his clothing also was constantly wet. On February 27th, 37 oz. of urine were collected; there was a trace of albumen. He subsequently had an attack of diarrhœa, and the quantity of urine which could be collected on March 1st and 3rd was only 20 oz.; it contained a trace of albumen on both occasions. There was irregular fever, great thirst, and the child looked very ill. On March 5th Mr. R.

W. Parker made an examination under chloroform, and withdrew one pint of urine; the stream only flowed when pressure was made on the abdomen. Eventually the abdominal tumour completely disappeared. No stone was found on sounding.

The patient did not micturate on either March 6th or 7th, and the bladder was again found to be as full as before. On March 13th a catheter was again passed, and 23 oz. of urine was withdrawn; he had micturated three hours earlier. The amount of albumen in the urine increased, and on March 16th blood was present; no casts. On March 17th very little urine was passed, and the bladder was not much distended; the blood in the urine had been replaced by pus. He died at 8.20 a.m. on March 18th.

Post-mortem examination.—On opening the abdomen the bladder was found to be much distended, but pressure with the hand caused it to be readily emptied by the urethra; on then inspecting the pelvis it was seen that the rectum occupied the middle line, and that on each side of it lay what appeared to be a portion of small intestine, but what was in reality the much distended and convoluted ureter; the two ureters presented precisely the same aspect. The bladder, so soon as the pressure on it was relaxed, became again immediately full. Pressure on the bladder caused regurgitation into the ureters at the renal pelves. The kidneys were large, and the pelves presented the appearance of having been much distended. The left kidney measured *in situ* five inches, and the right four and three quarters inches, and both extended close down to the iliac crest. The ileum crossed the right ureter at the brim of the pelvis one and a half inches before opening into the cæcum.

Both kidneys were greatly enlarged, chiefly owing to expansion of calices. The pyramids projected into the pelvis, and the medullary substance reached the surface opposite the apices of the pyramids; the cortical substance occupied the periphery in the intervals between the pyramids.

The left kidney substance was firm and pale, and the distinction between cortical and medullary regions was not well made out. The capsule was easily detached. The right kidney contained numerous whitish streaks in the medulla and numerous yellow-white nodules in the cortex, and on the surface. The capsule was thickened, and very easily detached; between it and the kidney-substance there were scattered collections of grumous fluid.

The bladder was large, and its wall was much trabeculated. There were numerous hæmorrhages into the mucous membrane. On the surface, and partly detached, were scattered greyish nodules. The ureters were, as has been said, much dilated, convoluted, and had in the convolutions a distinct fold of peritoneum resembling a mesentery; their mucous membrane was smooth and apparently natural. The opening of the ureters into the bladder was large, and admitted a cedar pencil. The other organs presented no abnormal appearances.

Microscopic examination of the left kidney showed fibrotic changes; the collecting tubes were large, so that a transverse section of the pyramids presented the appearance of a coarse network. The right kidney presented less fibrotic change, but contained numerous medullary abscesses, as well as elongated purulent collections parallel with the tubes of the pyramids. The large size of the collecting tubules was very noticeable in this kidney also.

Remarks.—This case is believed to be an example of congenital hydronephrosis (so called). The dilatation and the trabeculation of the bladder, together with the presence of fibrotic changes in one kidney (left), might appear to favour the view that the abnormality was produced during life. On the other hand there was a complete absence, both from the history and *post-mortem* appearances, of any cause of obstruction. The ease with which the urine contained in the dilated pelves, ureters, and bladder could be caused to flow from the first to the last-named organ was a striking feature at the necropsy, and there was certainly no urethral stricture nor phimosis. The normal valvular arrangement of the ureters in the wall of the bladder did not exist in this case, and the dilatation and hypertrophy of the bladder may be traceable to this, since the bladder must have been subject to the dilating force of the column of fluid as high as the kidneys, and had to contract against the same source of increased pressure.

In the second place, the anatomical characters of the abnormal parts, which included an unusual size of the collecting tubes, seems to favour the theory of an error of development.

The ureter and the collecting tubes of the kidney are developed from a *dorsal outgrowth* of the hinder part of the Wolffian duct. The outgrowth from the Wolffian duct grows forwards, and extends along the outer side of a mass of *mesoblastic tissue*, which lies mainly behind the dorsal aspect of the Wolffian body. This mass

of mesoblastic cells may be called the *metanephric blastema*.[1] This metanephric blastema forms the greater mass of the kidney tubercles, while the connecting tubules grow out from the ureter, and become continuous with masses of cells of the metanephric blastema, which become differentiated into the secreting tubuli.

November 15*th*, 1887.

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3. *A case of calculi contained in cysts in the kidney.*

By H. W. G. Mackenzie, M.B.

The specimen is from a woman, aged 69, who died in St. Thomas's Hospital within a few hours after the onset of cerebral hæmorrhage. No facts regarding her previous history were obtained. At the *post-mortem* the hæmorrhage was found to have occurred in the right internal capsule, and thence to have extended into the lateral ventricles. The arteries at the base of the brain and the aorta were atheromatous and calcareous, the aortic and mitral valves were thickened, and the left ventricle was considerably hypertrophied. Cysts were present in both kidneys, and the cysts in the right kidney, which is the present specimen, contained calculi.

The calculi, which are shown mounted separately, are all quite small, by far the largest weighing not more than five and a half grains, while the others, numbering some thirty, weigh only eight grains. The largest is of an oval shape like a small bean, its dimensions being about half an inch by one third by one quarter. The small ones are irregularly rounded or facetted and polyhedral, and average about one eighth of an inch in diameter. Their surfaces are smooth, highly polished, and of a grey colour, and in some cases marked with striæ. Dr. Bernays, who kindly chemically examined one of the calculi, reported that it was extremely hard, and that it consisted of pure oxalate of calcium.

The kidney is not enlarged, and beyond the presence of the cysts, which can be seen in the section and beneath the capsule, there is nothing which calls for notice. In the section it can be

[1] Balfour's 'Comparative Embryology,' vol. ii, p. 595 (1881). Conf. A. Sedgwick, "On the Development of the Kidney in its Relation to the Wolffian Body in the Chick," 'Quart. Journ. of Micros. Science,' vol. xx, 1880.

seen that the cysts are quite small, with the exception of two, one of which is in the cortex, and in the recent condition projected on the surface, and was about one inch in diameter, while the other is in the medulla not far from the first, and is about half an inch in diameter. The former contained the largest of the calculi, and the latter a good many of the small facetted ones. Most of the smaller cysts contained one of the small calculi. No communication could be made out to exist between the larger cysts and the pelvis, although such may possibly exist in the case of some of the smaller cysts. There were no calculi in the bladder, ureters, or pelvis.

The presence of cysts in the kidney is far from being uncommon, but the formation of calculi in the cysts is of very rare occurrence. I can find no record of a similar case having been brought before this Society. The only reference to such a condition which I have come across is in Dr. Dickinson's work on Diseases of the Kidney. It is there stated that in the Guy's Hospital Museum there is a kidney the cortical part of which contains a cyst the size of a hazel-nut, in which are some small calculi. The cyst, Dr. Dickinson says, has a narrow communication with the pelvis, and has probably been formed by an obstruction in the straight tubes and accumulation behind.

What determined the formation of calculi in such a case as this, and why they were present in so many cysts at the same time, it is impossible to say. The calculi were not the cause of the cysts, as the other kidney, while containing cysts, had no calculi. The formation of calculi such as these must have been a very gradual one. It is generally agreed that cysts in the kidney are the result of obstruction of tubules. To explain the formation of calculi in the cysts, it seems clear that either the obstruction of tubules was incomplete or that the cysts after their formation communicated with unobstructed tubules, so as to allow some flow of urine through them. Ebstein, in 'Ziemssen's Cyclopædia,' says that in some few cases oxalate of lime has been observed in the substance of the kidney, and that such a condition has been reported by Crosse and Meckel.

It might at first occur to one that, as was held at one time by Virchow, the cysts might be caused by the obstruction of tubules by minute concretions of oxalate of lime, that in some cases the obstruction was not complete, and that in these the minute con-

cretions developed into calculi. But there are several reasons for thinking this explanation improbable: first, the commonness of cysts in the kidney, without any evidence of concretions; second, the absence in this case of calculi in the other kidney; and third, the fact that obstruction by a concretion is much more likely to be complete than that due to some alteration in the wall of the tubule, and is on that account unlikely to favour the formation of a calculus. *April 3rd*, 1888.

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4. *Cystic degeneration of kidneys.* (*Card specimen.*)

By H. Montague Murray, M.D.

Plaster casts of kidneys, with a portion of the left, taken from a woman, aged 58, who had suffered from symptoms of granular contracted kidney for more than a year. The heart was hypertrophied, pulse hard, urine copious, with low specific gravity and a little albumin. Abdomen was large and prominent, and the walls very thick. No tumour was suspected during life. Thrombosis of left femoral vein occurred three days before death, and gangrene followed. Patient died with uræmic coma.

Autopsy.—Heart 17 oz. Kidneys, right 4 lbs. 9 oz., left 8 lbs. 4 oz. Each consisted of an enormous mass of cysts varying in size only. The largest is more than six inches in diameter. (The casts were taken the next day, when the cysts had already begun to shrink.) The walls of the cysts were thin and transparent, and the contents pale straw-coloured, and so clear that the subjacent cysts were distinctly visible. The connecting stroma was bright pink. The fluid was not gelatinous, alkaline, contained 4 per cent. of urea, and plenty of albumin. Granular débris and cholesterine crystals formed the only deposit. Arteries, veins, and ureters were normal. The cysts could not be injected from latter. On section distinct traces of kidney substance could here and there be found. Microscopical examination of this showed an enormous but very variable increase in the interstitial tissue as well as a number of cysts hardly visible to the naked eye. The cysts were lined with cub ical epithelium. *March 20th*, 1888.

5. *Hæmorrhage into a renal cyst.* (*Card specimen.*)

By Leopold Hudson.

The organ was obtained from a man, aged 39, who had been under the care of Dr. Cayley in the Middlesex Hospital, and died of endocarditis and chronic nephritis.

The right kidney, which weighed 6½ oz., was somewhat adherent to the surrounding perirenal fat. At its removal the upper end was seen to be much enlarged, and beneath the capsule was a yellowish-grey rounded prominence, semi-fluctuant, and looking much like new growth. The renal capsule was thickened and adherent over the whole organ. On section, it was found that the prominence was the external part of a large spherical cyst which had completely destroyed and replaced the renal tissue at the upper part of the kidney. It was limited by a distinct fibrous capsule, which externally was firmly attached to the proper capsule of the kidney, and internally abutted on, and was covered by, the mucous membrane lining the infundibulum of the ureter. This capsule could be separated by peeling from the kidney substance. The contents were of the consistence of soft mortar, and varied in colour in different parts from yellowish grey to deep brown; in places it had liquefied into a tenacious fluid of a dirty-brown colour. Microscopically this material was found to be an old hæmorrhage; it showed disintegrating blood-corpuscles, hæmatoidin, and cholesterine crystals, pigment, and fat granules, and amorphous detritus. The cyst being emptied it was found to be lined internally by a shining membrane, but no vessel could be discovered leading into it. The surface of the kidney elsewhere was granular and studded with numerous small cysts, none being larger than a pea. Their contained fluid was in some clear and limpid, in others viscid and of dark colour. The cortex was indurated and wasted, the medulla congested, and the renal pelvis contained a quantity of fatty tissue.

The left kidney was in a condition of advanced interstitial nephritis, its cortex containing many cysts, two being as large as a hazel-nut. The heart exhibited deformity and incompetence of the aortic valve, with consecutive hypertrophy and dilatation of

the left ventricle. There was also extensive and advanced aortic atheroma, right hydrothorax, pulmonary œdema, "nutmeg" liver, and engorgement of the spleen. *December 20th*, 1887.

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6. *Necrosis of the pyramids of one kidney.*

By F. Charlewood Turner, M.D.

In this kidney all the papillæ, with a great part of the pyramids, have become necrosed, and the sequestra had become detached from the living tissue to a greater or less extent.

One sequestrum is well seen in the upper part of the median longitudinal section of the organ. It consists of diverging tracts of tissue which correspond with the pyramids converging to the calyx. They are completely detached towards the papilla, in the apex of which they are united together, but they are still connected with the renal tissue at their distal extremities, where they spread out in finger-like processes corresponding with recesses in the basal part of the pyramids, and extending up to, or to within a short distance of, the deep surface of the cortical tissue. Where the sequestrum has become detached a cavity has been formed of corresponding shape with even walls, and the contiguous renal tissue appears unchanged as though it had been simply excavated. The cavities are larger than the sequestra separated from them, probably from shrinking of the latter in spirit. In the other calices similar partially detached sequestra are seen.

The kidney appeared otherwise normal. There was some injection of the mucous membrane of the pelvis of the organ but no lymph upon it. The other kidney and the bladder were normal.

The organ exhibited is the left kidney of a very obese female aged 60, who was admitted into the London Hospital under Mr. Tay on January 27th, 1888, with dry gangrene of the lower third of the right leg and foot, and died February 20th. She stated that the gangrene of the foot appeared one night, her throat being bad at the same time, and that she had applied first to the Throat Hospital in Golden Square on account of very extensive thrush on

the palate and gums. The urine was found to contain 2 oz. of sugar and was rather above the normal amount (60 to 75 oz.). She had had symptoms pointing to diabetes mellitus for at least several weeks. A note of the surgical registrar, Mr. J. Hutchinson, jun., states that "there was no retinitis. Her temperature was normal until the last five days of life."

The autopsy showed much atheroma of the abdominal aorta with areas of superficial erosion of the lining membrane, but without any adherent coagula. There were patches of atheromatous swelling in the thoracic aorta. The right posterior tibial artery was closed by a firmly adherent fibrinous plug a short distance below the bifurcation of the popliteal. In the latter vessel was a partly decolourised and slightly adherent coagulum. The heart weighed 9 oz. There was much adipose tissue over the right ventricle with some fatty infiltration of the myocardium, which was flaccid. The lungs were œdematous, the liver fatty, the brain and spinal cord apparently normal. In the velum interpositum were a number of chalky-looking bodies, which contained numerous concentrically laminated spherical bodies, like psammomata. On the right buttock were some large blebs containing blood-stained serum.

Necrosis of the pyramids of the kidney, independently of suppurative nephritis or pyelo-nephritis, is not described amongst the recognised lesions of those organs; and I cannot find any record of such lesions in our 'Transactions,' excepting in two specimens which I exhibited to the Society in 1885 and 1886,[1] which I may briefly allude to. The first was from a sailor, aged 34, who died with empyema and old phthisical lesions in the lungs. He had also tubercles in his lungs, and tubercular peritonitis with ulceration of the ileum, and lardaceous disease of liver, adrenals, and kidneys. The apices and central parts of the papilla of the pyramids of both kidneys were necrosed, shrivelled, and black, and partly detached. The second specimen was from a girl, aged 13, who died with valvular disease of the heart and recent endo-pericarditis. In the right kidney were softened and partially separated sequestra in the substance of several of the pyramids. The other kidney was unaffected. In neither organ, nor in the spleen, were there any wedge-shaped infarcts or necroses at the surface.

The simultaneous occurrence of necrosis in several or in all

[1] (1) 'Trans.,' vol. xxxvi, p. 268; (2) 'Trans.,' vol. xxxvii, p. 290. Both specimens are preserved in the London Hospital Museum.

of the pyramids of one or both kidneys in these cases can, I think, only have resulted[1] from thrombotic obstruction of the arterioles of the parts, the obstruction of the arteries being referable to lesion of their walls under the strain of the blood pressure, owing in part to deterioration of the general nutritive state of the body, and in part to influences interfering with the nutrition of the organs or causing an increase of the arterial blood pressure in them. The exceptional occurrence of the lesion is an evidence of the exceptional nature of the conditions by which it is determined.

The processes of softening and separation of the sequestra in these cases, contrasted with the processes of absorption and cicatrisation by which the wedge-shaped embolic or thrombotic necrotic tracts in the cortex are removed, are, I think, sufficiently accounted for by the condition of general nutritive defect indicated by the clinical phenomena both in the present case and in the first of those referred to, and more especially by the contact of the watery urinary secretion with the necrotic tissue. In none of them was there evidence of irritative or inflammatory reaction.

May 15th, 1888.

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7. *Kidneys from a case of renal calculus.* (*Card specimen.*)

By Frank Penrose, M.D.

The kidneys from a stunted, half-witted girl, aged 9½, who died from suppression of urine.

Left kidney and pelvis of ureter constituted practically a bag full of stones and putrid pus. The capsule is distinctly thickened, rather thicker than the cortex of the kidney, and the two together are not one sixteenth of an inch in thickness.

The calculi are about twelve in number, mostly very irregular in shape, and when moved one upon another *in situ* give a feeling almost exactly like surgical emphysema.

The *right kidney* is apparently congenitally small, consisting of

[1] Like the gangrene of the foot and leg with which they were associated in the present instance.

three lobes only, but in each of which the cortex is very much thickened.

History given.—Small baby, never talked, walked at three years, habits clean, fairly observant, and very mischievous. At five years old had a sharp attack of pain in *left* side, after which she passed a calculus *per urethram*. Pus in urine for next two years. At eight years old began to waste. Admitted to Hospital for Sick Children under Dr. Sturges in August, 1887, for cystitis.

On admission.—Passing large quantities of alkaline urine containing pus and ropy mucus. After washing out bladder with quinine for three weeks, became acid and free from pus and mucus. Child improved.

In middle of September began to vomit, have diarrhœa and a hectic temperature, resisted all examination, was evidently tender down left side and over bladder.

Urine-acid contained pus and granular casts. Child got gradually worse and died on October 6th of suppression of urine.

A week before death a feeling resembling surgical emphysema was felt in left loin, but its significance was not appreciated.

December 20th, 1887.

8. *Ulceration of bladder.* (*Card specimen.*)

By John H. Morgan.

The patient from whom this specimen was removed was a man, aged 50, but looking considerably older, who was admitted under my care on April 7th on account of retention of urine which had existed for twenty-four hours. He stated that two years ago he had passed blood in his urine, and that micturition had been painful, but after attending as an out-patient at the hospital the symptoms had passed off and the urine had become clear. About Christmas he experienced pain in passing water, micturition was frequent, and the urine contained blood whenever it was passed.

On admission, it was found that the bladder dulness extended to the umbilicus. No instrument could be passed into the bladder,

and an aspirator needle was introduced above the pubes, by means of which 24 oz. of dark brown urine was drawn off. Examination *per rectum* discovered some enlargement of prostate. Next day a silver catheter, No. 6, was with difficulty introduced *per urethram*, and 12 oz. of urine were withdrawn, deeply blood stained. This was allowed to remain in for two days, and the urine, still largely mixed with blood, continued to flow through, and by the side of, the instrument. Three days after its withdrawal the patient died.

Post-mortem examination.—Most of the viscera were found healthy, but the kidneys showed great dilatation of their pelves and diminution of the cortex. The bladder was filled with blood-stained urine. At the base, just behind the neck, was a large ulcer three by two inches, the greatest diameter being transverse. The edges of this were thickened and everted, the floor was rough. There was a small ulcer about the size of a threepennypiece of the same character, situated an inch to the left of the former. In the urethra was a large abscess cavity opening on the left side just in front of the membranous portion, and a second ulcer, three quarters of an inch long, was situated two inches farther anteriorly.

Microscopic examination.—At one part of the outer portion of the section numerous columns of cells were seen to be dipping in towards the subjacent (submucous) tissue. The cells in these columnar processes are flattened although some in the deeper layers are more rounded. In parts numerous typical cell-nests can be seen and other larger collections of cells surrounded by fibrous tissue. These latter cells are rounded or angular in shape, having central, rounded, or elongated nuclei plainly visible. The fibrous tissue is in very great quantity, and the elongated nuclei are plainly visible all over the field. In the deeper portion of the section numerous bands of involuntary muscle-fibres are to be seen embedded in the fibrous tissue. *May 1st*, 1888.

9. *Sloughing of the superior fundus of the bladder following cystitis and stricture.*

By W. Bruce Clarke.

Thomas W—, aged 36, was admitted into St. Bartholomew's Hospital on August 12th with retention of urine, which had lasted for three days, during the whole of which time he had scarcely passed any water; and though repeated attempts had been made to pass a catheter they had not been attended by any success. His stream had been getting smaller lately, but he had never had any instrument passed until his attack of retention came on.

The bladder dulness reached up to beyond the umbilicus. After a great deal of trouble, a small catgut was passed though a railroad catheter, and was retained in the bladder. By the next morning about three to four pints of urine had gradually drained away, and the bladder only appeared to reach to about midway between the umbilicus and the pubes. A good deal of blood passed with the urine, and as the contents of the bladder seemed to be more than usually solid and firm, it was conjectured that there might be a considerable amount of blood-clot.

Three days later a No. 7 English black catheter had been introduced, and was tied in. The urine that came away was deeply blood-stained, at times almost coffee-coloured, and there was a good deal of pain over the pubes.

Two days after this, and five from the time of his admission, the urethra would admit a No. 10 English, but he had no power of voiding his urine voluntarily, and it presented much the same colour and appearance.

Up to the end of August he remained much in the same condition. The urine was deeply tinged with blood and varied but slightly from day to day. The urine was foul and ammoniacal, 1018 sp. gr. and alkaline, and notwithstanding that the bladder was washed out four or five times daily with acetic acid, or boroglyceride, &c., it showed no tendency whatever to improve.

In September, when I went away for my holiday, the case came under the care of Mr. Baker, and as the man seemed to be getting

steadily worse, and his temperature rose for some days to 103° at night, perineal cystotomy was performed on September 7th.

The interior of the bladder was roughened, ulcerated on the surface and covered with phosphates.

For a few days the patient showed some considerable signs of improvement, but as great quantities of pus seemed to be coming from the bladder it was again explored a few days later, and a larger tube inserted.

He became gradually worse, and died on the 8th of October.

Post-mortem.—On opening the abdomen the cæcum was found to be adherent to the upper surface of the bladder, and on lifting it up the whole of the upper extremity of the bladder came away with it in a gangrenous condition. The adhesions between the bladder and the cæcum were very slight in character and recent apparently in origin, so that the two organs were easily separated from each other. There was a large slough of mucous membrane inside the bladder, and between the bladder and the rectum a considerable amount of pus, which was shut off from the general peritoneal cavity by adhesions.

The prostate was natural. The urethra had sloughed round the aperture of the wound. There was no stricture discoverable. All the viscera except the kidneys were healthy, and they were white and in a condition of tubal nephritis.

Observations.—Cases in which small sloughs occur in the mucous membrane of the bladder, or which may even involve all its coats, are not very uncommon in connection with disease or injuries of some portion of the central nervous system. In pregnant women too, sloughing may take place from pressure, or immediately in consequence of an acute attack of retention of urine, but I have been unable to find any case which is an exact parallel of the above. In this instance the bladder apparently was paralysed by the acute attack of retention. It never again regained its power of contractility, and in spite of all the efforts which were made to keep it sweet and to empty it at regular stated intervals, the process of inflammation eventually terminated, after just two months, in gangrene of a large portion of the viscus, and thus terminated the existence of the patient. *May 15th*, 1888.

10. *Notes upon the macroscopy of villous papillomata of the bladder.*

By E. Hurry Fenwick.

The incandescent-lamp cystoscope has greatly increased the value of naked-eye appearances of the pathological bladder, and especially of the various tumours which affect that viscus. There is no class of vesical tumour, except the fibromata, which so urgently demands or so amply repays surgical interference as the villous papillomata; and the electric light seems destined not only to afford us means of diagnosing the presence of such growths but also to indicate the form of operation which is the most suitable for their removal. It is therefore of the highest importance to ascertain from museum specimens whether these tumours are most often multiple or single, and what are the characters and localities of their implantation. Before entering upon these questions it is advisable to lay especial stress upon a variety of villous papillomata which may be as easily overlooked by the surgeon[1] as the pathologist. I refer to the 'subvilloid," "cropped villoid" growth, or papillôme en Nappe.[2] In this disease there is no definite clump or clumps of sessile or pedunculated growth, but patches of the mucous membrane are converted into a velvety condition by very short, closely-set, isolated papilliform projections. "Cropped villoid" exists in 18·3 per cent. of all the specimens I have examined of "villous" diseases of the bladder. There are eleven such to be found in the museums of England and Scotland. Most of them appear to have been overlooked, judging from the catalogues describing the bladders in which the morbid growth existed. This disease is, I believe, the direct outcome of some abiding source of irritation, such as a foreign body or residual urine. A good example, which Mr. Targett has kindly lent me from Guy's museum, is before you.

This bladder was shown to this Society by Dr. Rake[3] with other viscera which had been removed from a patient the subject of leprosy and syphilis combined. In examining the bladder, I found situated towards the apex a patch of subvilloid growth, which had been apparently induced by the irritation of a phosphatic-coated

[1] Keyes, 'Journal of Genito-Urinary Disease,' July, 1887, p. 247.

[2] *Vide* case, 'Pathological Trans.,' vol. xxxvii, 1886, p. 311.

[3] Dr. Beaven Rake, 'Pathological Transactions,' 1886, p. 545, vol. xxxvii.

straw, four and a half inches long. There is no history as to the introduction of this foreign body, but it was found with one end projecting into the prostatic urethra and the other extremity impinging upon that part of the bladder where the patch of disease is placed.

In this specimen (Glasgow Royal Infirmary, Ser. vii, 64), which Dr. Newman, of Glasgow, has kindly permitted me to exhibit, I found a small single patch of sub-villoid growth at the upper part of the bladder. It is situated upon the posterior wall and is about the size of a sixpenny piece. This large oval, uric acid calculus was found in the bladder after death. Its upper rim probably rested upon, and as probably produced, the patch I have just alluded to.

Other specimens are to be seen in the Museum of the Royal College of Surgeons and in the collections at Norwich, Liverpool, and Manchester.

In four of these the patch is small and situated towards the apex. A calculus was present in two cases and a foreign body in the others.

In five of these specimens the patch was placed upon the trigone, growing in four of these around the base of a benign tumour, and in the fifth around the orifice of a fistulous opening into the bladder.

In two specimens the entire posterior wall was carpeted with the growth. I have lately been able to diagnose such a growth by means of the electric light and successfully to remove it by scraping through the perineal route. Hæmaturia had lasted seven years.

Statistics.

Multiple villous papillomata occurred in 40 per cent. of the cases, the number of growths actually present in the bladder varying greatly. Thus:

2	tumours	were present in	3	cases
3	,,	,,	3	,,
4	,,	,,	1	,,
5	,,	,,	1	,,
8	,,	,,	1	,,
9	,,	,,	1	,,
22	,,	,,	1	,,

And many tumours (uncounted) in 9 cases.

Thus, in 17 cases out of 21 more than two tumours were found, a record which proves how diligent a search ought to be made in all cases where more than one growth is discovered by the operator.

The following conclusions have been drawn from a consideration of sixty specimens of vesical villous papilloma which I have examined in the various museums :

1. Papillomatous growths that are *single* are found in the inferior zone of the bladder in 86 per cent. of the cases.

2. These growths spring from the margins of the trigone or orifices of the ureters ; it is rare to find a growth actually originating upon the trigone.

3. These single growths are usually pedunculated or tend to become pedunculated. 43 per cent. are lightly pedicled, 20 per cent. are broadly pedicled or " sub-sessile," 33 per cent. are sessile.

4. Pedunculated villous papillomata are attached to the orifice of the right ureter in 43 per cent. of the cases, to the left orifice in 26 per cent., and to the interureteral bar in 10 per cent.

5. Pedunculation is more characteristic in the base than towards the apex of the bladder.

Certain rare positions for villous papilloma are to be noted. A few specimens exist in which definite villous tufts spring from the margins or base of those tiny dimples, saccules, or herniæ which are found in every grade of development in fasciculated bladders, but such a position is unusual. I have only seen one specimen of a villous papilloma arising in and sprouting from a distinct pouch (St. George's Hospital, 41^{A}). The growth in that specimen is the size of a walnut and projects from a pouch situated on the left side of the middle zone of the bladder at its antero-lateral angle.

March 8th, 1888.

11. *A fibroma coexisting with carcinoma of the urinary bladder.* (*Card specimen.*)

By E. Hurry Fenwick and Boswell Watson, M.D.

This bladder was removed from the body of a gentleman, aged 61, who had been under the care of Dr. Watson. Prior to the onset of symptoms, which occurred six months before death, the patient had enjoyed the best of health. The family history was good. A sudden and profuse hæmaturia was the first symptom noticed. The bleeding was quickly subdued by rest and gallic acid, but proved recurrent. The colour of the urine was usually of a chocolate shade, having many dark cylindrical clots mixed with it. Vesical irrigation with alum eventually controlled the bleeding. Cystitis ensued, and the patient gradually sank. No symptoms of carcinoma of the stomach were noticed.

Post-mortem by Mr. Fenwick.—Body very emaciated. On opening abdomen but little subcutaneous fat was found. The intestines were thin and empty, there were no signs of peritonitis, chronic or acute, yet the left lobe of the liver was intimately adherent to the anterior surface of the stomach. The bladder was evidently thickened, but no growth could be felt within its cavity.

Detailed examination.—On opening the bladder 2 oz. of puriform urine mixed with shreds of necrotic tissue and phosphatic débris were evacuated. The walls of the bladder were thick but not markedly hypertrophied. The cavity was contracted to the capacity of 4 oz. The mucous membrane showed signs of a severe form of chronic cystitis. Its surface was sprinkled here and there, especially the posterior surface, with a number of millet-seed-sized to pea-sized patches of yellowish white deposit distinctly carcinomatous in character. The colour of these growths marked them off very abruptly from the dull red mucous membrane around. Springing from the upper lip of the right ureteral orifice was a cherry-sized, slightly pedicled, oval-shaped tumour, which on section proved to be firm and white but did not exhibit any appearance of carcinoma. The prostate was absolutely healthy, and small for the age of the patient. The right kidney was half its natural size, and a section of it showed that very little renal structure remained. It had been converted, obviously by backward pressure, into a loculated sac with

a half-inch thick wall of renal substance. The right ureter and pelvis were correspondingly dilated. The left kidney and ureter were healthy, the former much hypertrophied (compensatorily). A large deposit of cancer existed in the left lobe of the liver, and in the outer wall, if not the mucous membrane, of that part of the stomach to which the liver was attached. The right lobe of the liver contained innumerable similar deposits of greater and lesser sizes. The lungs were œdematous, the heart was thin, and its muscle friable. The microscope showed the mass in the liver to be carcinomatous. The deposits in the mucous membrane of the bladder were of a like nature, and were apparently, from their multiplicity and minuteness, a *secondary* deposit.

The small tumour, pedicled at the right ureteral orifice, proved to be composed of fibrous tissue containing here and there spots of carcinomatous material.

From a careful consideration of the facts revealed by the autopsy, it would seem that the small fibroma had been a latent occupant of the bladder for a lengthy period, and had exerted a fair amount of obstruction upon the right ureter, for on no other grounds could the extreme condition of chronic interstitial nephritis of the right kidney be explained.

Seeing, however, that secondary deposit of carcinoma in the bladder is of great rarity,[1] it might very reasonably be contended that the tumour at the right ureteral orifice was a carcinoma and formed the primary focus of the disease. This supposition, however, loses weight by the fact that *pedicled* vesical carcinomata are extremely rare.[2] Moreover, carcinoma does not affect the ureteral orifices as a site for initial deposit.

Allowing then that the miliary deposit in the mucous membrane of the bladder and in the small fibroma was secondary to the mass found in the liver, we have in this case not only a very rare condition but also a very important question raised. What produced or incited the secondary deposit? Did the inflammation of the bladder which occurred a few months before death form a predisposing cause for the vesical infection? *April 3rd*, 1888.

[1] The bladder is invaded by secondary deposits from other sources in 1·8 per cent. of all cases (*i. e.* 12 cases out of 632).

[2] *Vide* article on Carcinoma, p. 178.

12. *Sarcoma of the urinary bladder.*

By E. Hurry Fenwick.

After a careful examination of pathological specimens and the literature of the subject I cannot but agree with Gross that sarcoma of the urinary bladder has been more overlooked than is usually supposed. Gross[1] says, "It is quite certain, however, that many neoplasms reported as encephaloid cancer occurring before the fortieth year, and composed of small round-cells closely packed in a homogeneous, or finely granular, or imperfectly fibrillated intercellular substance are sarcomata, and it is highly probable that tumours have been included under polypoid and villous growths which were of sarcomatous tissue." As, however, sarcoma of the bladder is currently reported to be "rare," I have taken the opportunity afforded by Messrs. D'Arcy Power and Vincent Jackson's specimens to place the following statistics of fifty cases of sarcoma of the urinary bladder before the Society.

Age.—The disease is commonest before five[2] and after fifty years of age.

Sex.—Males are more frequently affected than females.

Situation.—In children the inferior zone of the bladder is most often affected. In adults, although the base is not infrequently the seat of the disease, yet the most common position is the posterior wall by or between the ureteral orifices.

Implantation and number.—In children the growths are multiple, polypoid, and pedunculated or subsessile. In adults the tumour is generally single and sessile, 10 per cent. only possessing pedicles.

Size.—The largest single growth was the volume of a double fist; it was attached to the anterior wall. The average size was that of a hen's egg.

Surface.—A villous covering was found in 20 per cent.

Composition.—Most frequently the growth was found microscopically to be round celled (34·5 per cent.), but the construction varied greatly in different parts of the same tumour. Spindle-celled sarcomatous tissue formed 17 per cent. of the specimens. In

[1] Gross, 'Diseases of the Urinary Organs,' p. 147, 1876.

[2] Vesical sarcomata in children has often been described as myxomata. believe sarcoma is much the more usual growth in children.

only one case was cartilage found.[1] Two cases are on record in which the surface of villous papilloma has been found degenerating into sarcoma.

An interesting and important specimen of sarcoma arising in a diverticulum was exhibited by Mr. Roger Williams in 1883 before this Society.

Since the above was written Mr. Southam has recorded six cases of sarcoma which had been under his own and his colleague's care at Manchester. His analysis of the cases is as follows:

Age.—9, 40, 46, 47, 60, 69 years.

Sex.—One female, five males.

Situation and connections of tumour.—Five cases were sessile and situated on the base or posterior and lateral walls. One case was pedunculated and placed upon the anterior wall on a level with the upper border of the pubes.

Surface.—In three cases the surface was distinctly villous.

Nature of tumour.—Three cases were composed of round-celled sarcomatous tissue, one of which was "alveolar" in parts, and another "lympho-sarcomatous" in places. Three cases exhibited mixed celled growth (round and spindle celled). The autopsy in three cases showed the growth to be confined to the bladder wall, and in one case only were secondary deposits discovered (*i. e.* in the kidney). *March* 20*th*, 1888.

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13. *A case of sarcoma of the urinary bladder.*

By D'Arcy Power, M.B.

Sarcoma of the bladder is a comparatively rare disease indeed. The ordinary English text-books on surgery hardly refer to it, whilst Gross in his 'System of Surgery'[2] says that "as verified by minute examination sarcoma is one of the rarest of the neoplasms of the bladder." In the following paper, however, I shall endeavour to show that this statement is hardly founded upon fact, or at any rate that my predecessors, the curators of the museum at St.

[1] Shattock, 'Pathological Trans.,' vol. xxxviii. p. 183, 1887.

[2] Vol. ii, p. 620, ed. vi. Philadelphia, 1882.

Bartholomew's Hospital, have been singularly fortunate in preserving a considerable number of specimens of vesical sarcoma.

The first example is a bladder which has been laid open along its anterior wall; its cavity is seen to be obliterated, except at its upper part, by the new growth which has infiltrated its anterior and lateral walls as well as the fundus. The portion of growth which occupies the cavity of the bladder is a tuberous and cauliflower-like mass. The rectum has become involved by its extension backwards, and near the anus the tumour actually projects into the cavity of the bowel. The projecting portion, however, has broken down, and a fistulous passage has in this way been established between the bladder and the rectum. A piece of catheter has been passed through an opening at the upper portion of the specimen which was made by supra-pubic puncture of the bladder, a fortnight before the death of the patient. The preparation was obtained from a man, aged 64, who had suffered from hæmaturia for eighteen months. The growth was partially removed by median cystotomy and dilatation of the prostate, but as it continued to increase in size, and micturition became difficult and painful, the bladder was punctured above the pubes. Microscopically the growth is a typical mixed-celled sarcoma.[1]

Four other cases of sarcoma of the bladder are preserved in the museum of St. Bartholomew's Hospital. In the first[2] of these the new growth is single and infiltrates the whole organ, except the anterior third, and nearly fills its cavity. The bladder is not hypertrophied, nor are either of the ureters dilated. The uterus is involved in the infiltration, but the vagina is free. Microscopically the growth consists of round sarcoma cells with little or no matrix.

In another specimen[3] the bladder has several masses of new growth, sessile on its mucous membrane. The largest of these masses is situated over the orifice of the right ureter, and infiltrates the muscular as well as the mucous coat of the bladder, whilst a smaller mass grows from the left side of the urethra. Numerous masses of growth, measuring on an average three quarters of an inch in diameter, are scattered over the rest of the mucous membrane. The bladder is slightly hypertrophied. It was removed

[1] It is preserved in the museum of St. Bartholomew's Hospital, Series xxx, No. 2419 (*a*).

[2] Ib., No. 2428.

[3] Ib., Series xxx, No. 2429.

from a man, aged 57, who exhibited symptoms of the disease for six months before his death. Microscopically the tumour is a mixed-celled sarcoma which is undergoing mucoid degeneration.

In a third instance[1] the bladder contains several firm round tumours which are situated between the mucous and muscular coats of the fundus. One tumour projects into the cavity of the organ, whilst the rest protrude on its outer surface, being covered by the peritoneum and the muscular fibres of the organ. Microscopically the growths consist of small round-cells embedded in a homogeneous matrix.

The last specimen[2] preserved in this series is perhaps the most interesting, for it is of the nature of an alveolar sarcoma. It is described in the catalogue as a bladder, laid open by a vertical incision through its anterior wall. A pedunculated growth is attached to its inner surface, stretching transversely across the fundus of the bladder, immediately behind the apertures of the ureters, which are much dilated. The mass is attached at either side, but free in the centre, and was so situated that it might lie forward over the urethral orifice, or be propelled in that direction when attempts were made to void the urine. The tumour, irregularly lobulated, consists of a fine filamentous structure scattered through a granular substance, and invested by a quantity of tesselated epithelium. The walls of the bladder are much thickened. At its upper surface the cavity of an abscess commences and extends to the umbilicus, but no communication can be traced between the two, although the urine continued to escape by the abscess up to the time of the child's death.

The small papilla close to the vesical termination of the abscess is all that appears of the urachus. A bristle passed some way down it, but could not, without violence, be forced into the bladder.

From a child who had suffered for eight weeks from extreme pain during micturition, presently followed by severe pain in the abdomen. A swelling formed about the umbilicus, softened, and was opened with a lancet, some healthy pus escaping. Urine began to dribble away from this opening, scarcely any escaping by the natural channel. The child, after lingering in a wretched state for some days, died.

I am enabled by the kindness of Mr. Hurry Fenwick to add the

[1] St. Bartholomew's Hospital Museum, series xxx, No. 2430.

[2] Ib., series xxx, No. 2419.

details of four additional cases of sarcoma of the bladder which are preserved in the pathological collections in London.

In the Hunterian Museum[1] is a lympho-sarcoma of the bladder from a man, aged 68. The entire organ is infiltrated, and so filled with the growth that only a small cavity is left at the trigone.

In the same collection[2] is a male bladder containing a single sessile growth situated on the left side of the orifice of the left ureter. The growth infiltrates the whole thickness of the vesical wall. It is probably a myo-sarcoma.

In the museum of Guy's Hospital[3] is a male bladder containing a mass of spindle-celled sarcoma situated near the right ureter. The rest of the mucous membrane is healthy. The growth consists of two parts, a body which is sessile and is covered with long villi, and an outer portion which is smooth, and which consists of looser tissue. The bladder is not hypertrophied, nor are the ureters dilated.

In the museum of the Middlesex Hospital[4] is a villous fibro-sarcoma which exists as a single oval growth attached to the bladder by a broad flattened base. The tumour is situated close to the entrance of the right ureter, and is of the size and shape of a hen's egg. It is soft and spongy in texture. As in the preceding cases the bladder was taken from a man.

The preceding cases show that there are at the present time in London nine cases of sarcoma of the bladder of which three may be classified as mixed-celled sarcomata and two as round-celled sarcoma. The remaining four are isolated instances of fibro-sarcoma, lympho-sarcoma, myo-sarcoma and alveolar sarcoma respectively.

March 20th, 1888.

[1] The Royal College of Surgeons, No. 3706.
[2] Ib., No. 3707 (*a*).
[3] Guy's Hospital Museum, No. 2104^{22}.
[4] Middlesex Hospital Museum, No. 1750.

14. *Sarcoma of the urinary bladder.*

By Thomas Vincent Jackson.

S. G—, aged 76, was first seen in September, 1885; is married and has a family of three daughters. A native of Switzerland, but since early manhood he has resided in England, and chiefly in Wolverhampton, and ever since he has pursued with much energy an active although not laborious, but yet at times anxious occupation, and looking back upon his past life from his present standpoint of three score and sixteen years his memory fails to remind him of any illness antecedent to the present one. Of his family history he knows very little, and that little seemed so unreliable that no note was made of it.

Mr. G— has consistently through life been most regular in his habits and has likewise been temperate in his eating and drinking. He is a non-smoker.

Naturally of spare habit, he states that physically he has undergone with increasing years little change, owing as he believes to his life-long activity, energy, and to the possession of a nervous temperament.

Two years since, much to his surprise and without any provocative that he remembers, he observed on casually urinating that blood was visible in the chamber. For a time it disappeared, then was observed again, but on each occasion the quantity of blood voided visibly increased until just before he came to see me it seemed as if only blood was expelled. The result of a careful examination was, that I arrived at the conclusion that the source of the hæmaturia was vesical, and for these reasons:

1. That the frequency of micturition by day and by night was very much increased, and that an occasional although not a constant symptom was pain at the end of the penis, at first only noticed at the termination of the act of micturition, but afterwards the pain was more continuous.

2. That on one occasion blood had in large quantities appeared in the urine after exercise.

3. That although the exploration of the interior of the bladder with a sound, as well as its bimanual examination by the finger in

the rectum and the fingers of the other hand pressing as deeply as was permitted above the pubes, supplemented by a careful qualitative and microscopic examination of the urine, yielded negative results, yet, as the annoying symptoms—hæmaturia and frequency of urinating—were much intensified for a few days by the internal exploration and the external manipulation of the bladder, it was, I think, a fair inference that the source of the blood flow was undoubtedly vesical.

I may state that the reaction of the urine was acid, and that it continued so until death occurred.

An exploratory operation was advised, but by himself and his family was at once opposed and ultimately declined. On February 28th, 1886, death took place from asthenia.

A limited *post-mortem* examination was made by Dr. Evans; the bladder after inspection only being removed. A growth was seen to occupy the base of the bladder, covering the trigone and extending forwards to the prostate, the posterior wall of the bladder being unaffected. The growth was single, and although not distinctly pedunculated yet the attached portion was circumferentially smaller than the free portion.

The microscopic examination was made by Dr. Heneage Gibbes, and the following is his report: "The tumour is made up of rounded cells, varying in size in different parts, and a fibrous stroma. Throughout it runs a large number of blood-vessels in direct contact with the cells. In some parts the cells are small and densely packed, and the stroma can only be made out with difficulty; in other parts the cells are larger and the stroma surrounds each cell; other parts are intermediate between these two conditions. The blood-vessels are very numerous and irregular in calibre; in many places they are much dilated and full of blood. In other parts there is a denser plexus of capillary vessels, and the interstices of the surrounding tissue are full of extravasated blood. The tumour is of a sarcomatous nature, varying in its structure in different parts. That portion containing the large round cells surrounded by stroma resembles what has been described by some writers as alveolar sarcoma. In other parts it resembles a round-celled sarcoma of the ordinary type but differs in being much more vascular than they usually are. In the most delicate parts the stroma somewhat resembles that found in myxomata."

Remarks.—Sarcomata are admitted to be the least frequent of the various kinds of tumours—innocent and malignant—which invade the bladder, therefore it is desirable that no opportunity should be omitted of examining and reporting any given example when the occasion is afforded.

The specimen which I exhibit to the Society to-night is not one of the most ordinary, for in its structure it is variable, in one portion resembling what has been described as alveolar sarcoma and in other parts a round-celled sarcoma (the most common kind).

Again, in persons of advanced or advancing age the growth more frequently affects the posterior wall of the bladder, and is not as a rule pedunculated or inclined to be pedunculated; but in the specimen now exhibited the base of the bladder is the seat of the disease, and instead of being sessile there is a pedicle, although perhaps a somewhat broad one.

Hæmaturia, as a rule, is not the first symptom in malignant affections of the bladder but is generally preceded by frequent micturition.

The manual determination of the presence of a sessile or semi-sessile sarcomatous bladder tumour will often be a difficult problem, a hit or miss kind of business, and the certainty of its presence will be quite as much a mere impression as a positive demonstration.

The question is still, I think, undecided whether a sarcomatous tumour when diagnosed, and moreover when felt or seen, should in persons of an advanced age be meddled with? What is now wanted is not the mere recital of the operation, explorative or complete, but the after-history of the case from that event to the end of life. *May 15th*, 1888.

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15. *"Contact" carcinoma of the urinary bladder.*

By E. Hurry Fenwick.

It is as yet a moot point in pathology whether malignant disease can or cannot multiply itself by implantation or inoculation. A certain number of facts appear to support the opinion that an

epitheliomatous ulcer can produce upon an apposing surface, or even upon one with which it is frequently brought into contact, a growth exactly similar to itself.[1]

Thus Kauffmann[2] relates a case of a woman who had cancer of the dorsum of the right hand, and subsequently of the conjunctiva of the right eye. Her relatives stated that she constantly wiped the right eye with the back of the right hand.

Lucke[3] had a case of ulcerated carcinoma of the edge of the tongue with inoculation of the mucous membrane of the cheek of the same side.

Cayley[4] showed a specimen to this Society of epithelioma of the posterior wall of the vagina, which had produced a similar growth on the anterior wall of the same canal, the patch thus inoculated being exactly apposed to the site of the initial lesion.

Dr. Alexander Williams, of Tarland,[5] reports a case of a cancerous tumour situated on the inner aspect and a little above the middle of the right thigh of a woman aged 60, producing apparently a similar growth on the corresponding part of the left thigh. Moreover, cases of epitheliomatous ulcers of the lip and labium pudendi have been encountered which have apparently induced, by contact, a corresponding growth in the opposing fellow fold.

Whilst examining 150 museum specimens of cancer of the bladder I was greatly struck with the isolation of the deposits of the growth. In some of these multiple vesical carcinomata the tumours were so obviously placed upon surfaces which would come into apposition on contraction of the viscus that I could not help being impressed with the appearance they presented of having been inoculated from a primary tumour.

These specimens, which I am able by kind permission of Mr. Shattock to demonstrate here to-night as illustrative of the subject, will show to some extent a point which is, of course, extremely difficult to establish.

[1] Richard Hall, "Etiology of Malignant Tumours," 'Annals of Surgery,' 1885, p. 457, vol. ii.

[2] Kauffmann, "Ueber multiplicatät des primären Carcinoms," 'Virch. Arch.,' Bd. 80, p. 347.

[3] Lücke, "Die Lehre von den Geschwülsten," 'Billroth und Pitha Handbuch,' Bd. 11, Abth. 1, p. 50.

[4] Cayley, 'Path. Trans.,' vol. xvii, p. 198, 1886.

[5] A. Williams, 'British Medical Journal,' December 17th, 1887, p. 1369.

Case 1 (St. Thomas's B. B. 36).—The bladder is cut open in front. The posterior wall is free; the anterior wall is free from projecting growth but densely infiltrated. Springing from apposed lateral surfaces are ulcerated masses of growth. The tumours of the two sides, however, differ; the right-sided mass is the larger. In vesical contraction its upper third would shelve above the tumour of the left side. The mucous membrane against which this over-riding piece would come is spotted over with carcinomatous tubercles. The sequence of events might have been this: The left-sided growth produced by irritation the tumour found upon the right; the growth of the latter being more rapid than the former it overlapped its forbear, and impinging on the healthy membrane above it produced a second generation of malignant cells.

Case 2 (St. Thomas's B. B. 25).—On the posterior wall of the bladder, immediately above the right ureteral orifice, is a small tumour somewhat like a flattened raisin. On the right side of the trigone upon which this subsessile growth would rest are a number of small nodules of growth.

Case 3 (St. Thomas's B. B. 31).—The primary growth appears to be the largest tumour which is pedicled in the left lateral zone. The apex of the bladder against which the growth would press is covered with secondary deposits. The base shows only marks of acute cystitis.[1]

In 10 per cent. of the cases of vesical cancer villous papillomatous growths coexist in the bladder with the carcinoma, apparently produced by irritation. It is with the object of drawing attention to this important point that I have broached the subject.

February 7th, 1888.

[1] Compare cases by Roger Williams, "Sarcoma in a Diverticulum," 'Path. Trans.,' 1883, vol. xxxiv, p. 152; Bruce Clarke, "A Case of Double Epithelioma of the Bladder," vol. xxxviii, p. 181; Heath, "Villous Sarcoma," 'Medical Times and Gazette,' Dec. 13th, 1879; St. Mary's Hospital, Hf. 4, Hf. 9; Guy's Museum, 2103[60]; Middlesex Museum, 1952; St. Bartholomew's Hospital, 2420.

16. *A case of carcinoma of the bladder.* (*Card specimen.*)

By E. Hurry Fenwick.

This bladder was removed from a patient aged 51. The history of the case is briefly as follows: Eight months before death the patient began to experience a constant desire to pass water, having to strain to complete the act. He suffered pain at the end of the penis after micturition. He also had pain in the hypogastrium and thighs, which was increased by locomotion. Intermittent hæmaturia was also noticed. He had been a free drinker.

Status præsens.—He is a well-built man, with a good deal of subcutaneous fat; the glands in the groin are hard and shotty as if syphilitic, those on the right side being especially enlarged. He is sent for the relief of a supposed calculus, but examination of the rectum shows that a hardness and unevenness exist at the base of the bladder and vesiculæ seminalis, which is very suggestive of carcinoma. This condition is especially marked on the right side. The prostate itself is not enlarged or hard. He was sounded, but with difficulty, the instrument having to be much depressed. He gradually sank.

Autopsy.—There is much visceral fat. The peritoneum is adherent to the back of the bladder in patches. On putting the hand into the pelvis the course of the vessels is felt to be marked out by large Brazil-nut-sized glands. The margin of the true pelvis is thus very uneven, and its opening is contracted. (The pelvic viscera were removed *en masse*.) Carcinomatous deposits are to be felt along the back of the bladder. These are evidently glands which follow the course of the vasa deferentia; they are seen to be in direct communication with the iliac chains, and the enlarged glands in the groin. The bladder is opened in the middle line, and the muscle wall is seen to be slightly but distinctly hypertrophied. Blocking up and distending the prostatic urethra is a firm mass of cancer, which has its origin in the right side of the trigone, but which extends upwards to implicate the right ureteral orifice. The size of the growth is equal to a small Tangerine orange. Its surface is lobulated and uneven; it cuts very firm. It is not ulcerated, except at one spot, where a small cavity is found plugged with a

recent blood-clot. Sections made through the prostate show it to be implicated in patches, but the disease has evidently commenced in the trigone and not in the prostate. The vesiculæ seminales are hard, their cavities are contracted, and their walls infiltrated with carcinoma. No deposits are to be found elsewhere.

April 3rd, 1888.

17. *Primary carcinoma of urinary bladder.* (*Card specimen.*)

By G. H. Makins.

The specimen is half of an ulcerated growth from the floor of the female bladder. The affected portion was roughly circular in outline, $2\frac{1}{2}$ inches in diameter; surface rugged and uneven at the margin; a number of small polypoid excrescences exist. Wall of bladder infiltrated and thickened to about three quarters of an inch. The external aspect shows a small adhesion to the cervix uteri, and also the termination of the left ureter considerably dilated and hypertrophied. In the recent condition the section of the growth was white in colour and in consistence hard and gristly.

The growth, of which a section was exhibited, consisted of a stroma enclosing large alveoli containing epithelial cells, resembling those of the mucous membrane of the bladder, but there were no "cell-nests."

The remaining portion of the bladder was hypertrophied, varying in thickness from a quarter to one third of an inch. The mucous lining was of a dark slate colour with a few patches of bright red; it was roughened and villous throughout, and in a few spots encrusted with phosphates.

The internal iliac glands were infiltrated, hard, white, and cupping on section. No further secondary growths were discovered.

Ureters moderately dilated and hypertrophied, especially the left, distended with pus, and red and inflamed throughout.

Kidneys: Right $6\frac{1}{4}$ oz. Pelvis slightly dilated, both it and calices full of pus; numerous small yellowish spots in cortex, some projecting from surface; capsule slightly adherent. Left $2\frac{3}{4}$ oz. Converted into a large branching cavity full of pus. Capsule adherent.

Uterus: Slight endometritis; cervix adherent over a small area to left part of base of bladder but not infiltrated.

Ovaries healthy; small points of adhesion between left broad ligament and bladder.

Liver firm.

Spleen normal.

Heart normal.

Lungs: Muco-purulent secretion in large- and medium-sized tubes; otherwise normal.

Pericardium and pleuræ normal. Body much emaciated.

Clinical history.—F—, aged 60; married, fifteen children; no heredity traceable; has always been healthy. Symptoms consisting of frequent micturition, accompanied by pain, scalding, and hæmaturia dated from Christmas, 1886. She was admitted into St. Thomas's Hospital on June 24th to have some urethral caruncles removed, which was done by excision and cauterization of base. She left on July 5th, but returned on August 10th with aggravated symptoms. She had passed a considerable quantity of blood as clots in the interval, and micturition, which was extremely painful, had been necessary every hour throughout the night. The urine contained numerous pus-cells and blood, but nothing distinctive of cancer.

On September 3rd the urethra was dilated and the bladder explored by the finger when the true nature of the case was discovered. A small polypoid growth from the margin of the ulcer was removed with the snare, and its nature was determined by microscopical examination.

The patient subsequently developed well-marked signs of suppurative nephritis and died on October 22nd, 1886.

December 6th, 1887.

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18. *Bilharzia hæmatobia, coexisting with an epitheliomatous growth of the bladder.* (*Card specimen.*)

By E. Hurry Fenwick, for Dr. Thomas Harris (Manchester).

This specimen represents the half of a bladder, which was kindly sent for the Owens College Museum by Mr. Reginald Harrison,

of Liverpool, as an illustration of a bladder affected with the bilharzia disease, but on microscopical examination it was found that, in addition to showing a very large number of the ova of the *Bilharzia hæmatobia* there was present a large epitheliomatous growth sprouting into the interior of the bladder, the cavity of which organ it had, judging from the half available for examination, almost entirely filled. The bilharzia ova were not present in the growth, but at its base,—a large number existing in the surrounding mucous and submucous tissue.

No history could be obtained of the case, except that the man was believed to have suffered for many years from the bilharzia affection, and the malignant disease was not suspected during life.

The case appears to be interesting on account of the possible connection of the malignant growth with the bilharzia affection. It is quite possible that malignant disease may have been occasioned by the irritation produced by the bilharzia ova, and that whilst usually the effect of the ova in the mucous membrane of the bladder is only to cause the production of an inflammatory granulation tissue, in this case the irritation had occasioned the development of an epithelioma. *May 15th*, 1888.

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19. *Urinary bladder and kidney from a case of Bilharzia hæmatobia.* (*Card specimen.*)

By F. S. Eve.

The bladder has been divided by an antero-posterior section. Springing from its floor is a flocculent fungoid growth. A larger growth projects from the much thickened anterior wall. Its cavity is contracted, and the mucous membrane in parts, ulcerated.

Microscopically, the fungoid growths were of the nature of inflammatory new-formations. On their surfaces were masses of eggs and egg-cases of the parasite.

The ureters are dilated.

The pelvis and calyces of the kidney are dilated, and its parenchyma extremely atrophied.

Ova were found in the capillaries of the lungs, kidneys, and skin.

Presented to the Museum of the Royal College of Surgeons by Dr. Milton, of Cairo. *February 7th*, 1888.

20. *Extreme tuberculous ulceration of the bladder; tuberculosis of the urethra.* (*Card specimen.*)

By Percy Kidd, M.D.

In addition to marked pulmonary phthisis the patient was the subject of extensive tuberculous pyelitis (the left kidney), tuberculosis of the right kidney, left ureter, prostate, vesiculæ seminales, left testicle and vas deferens, larynx and intestine.

The bladder, with the exception of a small portion of the right lateral wall, presents one continuous ulcerated surface, which is covered by a thin layer of whitish urinary salts. The walls of the bladder are only slightly thickened. The ulceration is superficial, its surface is coarsely granular, being marked here and there with bright red points, and at its borders there is a zone of dusky redness. A few small islands of ulceration of a similar appearance with a surrounding halo of congestion are seen in the unaltered mucous membrane just outside the margin of ulceration.

The ulceration extends into the urethra for about the distance of one inch, and farther on the mucous membrane is studded with groups of opaque miliary tubercles to within about half an inch of the meatus, which is unaffected. The prostate and vesiculæ seminales contain caseous masses, the left vesicula being much enlarged. The left ureter in its lower half is converted into a thick solid caseous cord.

The patient during life complained of no bladder symptoms. The urine contained some mucus, and a small quantity of albumin.

April 17th, 1888.

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21. *Imperforate urethra in a fœtus of about the fourth month.*

By Samuel G. Shattock.

[With Plate XI.]

The fœtus measures in the fully extended position, from the vertex of the head to the heel, 14·3 cm. As computed from Toldt's table, given in Spiegelberg's text-book of midwifery (trans-

lated by the New Sydenham Society, 1887), this will give an approximate age of slightly more than four months. The male sex is fully declared. Externally, beyond a great fluid distension of the abdomen, nothing unusual presented itself.

The umbilical cord is normally attached to the abdominal wall, and is itself normally constituted.

An incision into the distended abdomen reveals a large, smoothly-walled space occupying the pelvic cavity, and apparently that of the whole abdomen; it appears at first sight as though the abdomen were wholly devoid of viscera. The simplicity of the interior of the cavity is broken only by the presence of two paired openings 2·5 mm. in diameter at its posterior and lower part. The openings are 1·7 cm. apart, and lie ·6 cm. above the level of the symphysis pubis.

A systematic dissection shows the large cavity noticed to have a proper wall, distinct from and of about the same thickness as that of the abdomen. The abdominal wall, indeed, admits without difficulty of being peeled from the front and sides of the sac in question, which measures in the vertical direction 5·2 cm. ($2\frac{1}{16}$th inch), in the transverse 3·5 cm., and from before backwards the same.

On separating the sac from the deeper posterior parts, the paired orifices in its base are seen to be the openings of the lower ends of the ureters, both of which are equally dilated, and thrown, by reason of their increased length, into tortuous varicose coils.

The ureters open without obliquity by circular patulous orifices into what is therefore an enormously distended urinary bladder. The posterior surface of this dilated bladder presents faint transverse ridges like those due to the muscular hypertrophy seen in most cases of ordinary vesical or urethral obstruction.

The kidneys measure each, in the vertical direction, 1·5 cm., and are surmounted by a normal adrenal. Longitudinal section shows each kidney to be in the extreme degree cystic, and microscopic preparations offer admirable specimens of the condition known as general cystic disease, examples of which have been from time to time described in the Society's 'Transactions,' and are contained in most museums of any pretension.

The histology of the kidney makes it plain that the cysts are of the kind classed as retention-cysts. They form a series of spaces of various shapes in section; circular, oval, reniform, flask-shaped, or tortuous, irregularly-spreading cavities with lesser branches

DESCRIPTION OF PLATE XI.

Illustrating Mr. Shattock's specimen of Imperforate Urethra. (Page 185.)

From a drawing by himself.

S. G. Swancock, del.

F. Huth, Lith.r Edin.r

projecting from their sides. It is difficult, indeed, to find any normal tubuli in the renal substance. The cysts are lined with beautiful cubical epithelium. In nearly all cases the glomeruli project into cystic dilatations of their capsules, and it is observable that dilated tubuli open freely into the renal pelvis.

The stroma between the cysts, &c., is abundant, with ovally nucleated corpuscles; the larger trabeculæ are of looser texture in their more central parts, with branched corpuscles. The arterioles are healthy.

Above and behind the distended bladder lie the various abdominal viscera, closely pressed against the diaphragm and other walls of the abdomen; the thoracic viscera are normally developed. More particularly the intestine terminates in a well-formed rectum, which has a free conjunction with the anus.

Remarks.—Whatever may be the difficulty in certain cases of accounting for distension of the fœtal bladder and ureters, in the present the cause is obvious enough; there is complete atresia of the bladder.

There are, however, two or three debateable questions raised by this specimen. In the first place, is the imperforate condition primary and original, or has it occurred subsequently to a normal development of the parts? There is plain evidence adducible that diminution or closure of canals occurs at times during the process of development from secondary changes, setting aside the considerable class of clases in which this obliteration occurs as a normal event.

The complete closure, for example, of the duodenum at times observed in the fœtus, cannot be explained except as a secondary abnormal occurrence in the developmental process. Two cases of this kind are figured by Förster in his 'Missbildungen;' another is incidentally referred to by Spiegelberg (loc. cit., p. 462), and instances of the same have been recorded by Mr. Sutton in the 'Med.-Chir. Trans.,' and by Dr. Charlewood Turner in the 'Path. Soc. Trans.,' 1887. Again, some cases of congenital atresia of the pulmonary or of the aortic arterial aperture are clearly explicable on intra-uterine diseases of the valves. Dr. Peacock has recorded such cases ('Malformations of the Human Heart,' 2nd edit.), and some while ago I showed at the Society a specimen of complete atresia of the aortic aperture of a similar nature ('Path. Soc. Trans.,' 1881).

In the present case, however, the imperforate condition is most probably primary or original rather then secondary in its nature; and it may be looked upon as the counterpart of that which constitutes imperforate rectum. It is clear, on a moment's reflection, that if the hind-gut and the urogenital sinus be continuous in the manner and degree they normally should, when the cloacal depression lays open the one it will lay open also the other into a common space.

Nevertheless, there are cases of perforate urethra combined with imperforate rectum, and cases (such as the present) of imperforate urethra combined with perforate rectum. And it must be assumed, I submit, that in either class of case the original continuity between the gut and the urogenital sinus has been partially or completely closed, or has been from the first unduly small or unnaturally placed, before the parts are reached by the cloacal depression, and that either the gut or the sinus may be opened without the other.

It is of some interest to inquire whether the urethra is normally formed or not. There is a well-developed penis and scrotum and a distinct preputial orifice, and doubtless a urethra, if it would be right to so mutilate the specimen as to see; for I may supply this deficiency in observation, perhaps, by referring to a specimen of somewhat similar kind in the museum of Guy's Hospital. In this specimen (2551[47]), a fœtus of about the fifth month, the distended bladder has ruptured into the peritoneal cavity. In the recent state the bladder held about half a pint; it reached the prostate and there terminated. At the back of the bladder was a small ruptured opening.

The ureters are tortuous and dilated. The extravasation of urine has set up peritonitis; the abdomen, it is noticed, contained inflammatory lymph and serum (for serum understand probably urine).

The wall of the bladder is hypertrophied and fascicular. There is a normal anus through which the intestine opens.

Now, in this specimen there is a well-formed urethra in the body of the penis. The kidneys measure in this specimen 1·1 cm. in vertical diameter, as against 1·5 cm. in the younger subject of the present communication. Through Dr. Goodhart's kindness I have been able to examine them microscopically. A vertical transverse section a little posterior to the proper mesial plane of the kidney

shows a considerable degree of fibrosis, and the presence of irregularly-shaped retention-cysts lined with cubical epithelium and sparsely scattered in the renal cortex, some of them close beneath the capsule. If there is any dilatation of the pelvis it is inconsiderable.[1]

Theoretically the point of occlusion should be in the urogenital sinus anterior to (or below) the aperture of the common ejaculatory ducts. It is at this spot that the gut usually opens into the male urethra in cases of imperforate rectum complicated with recto-urethral fistula. The enormously distended bladder should therefore include the urogenital sinus; and the ejaculatory ducts should open into the lowest part of the cavity, but I have been unable to test this by dissection.

It will be seen, however, in the specimen that the lower portion of the sac, which fills the pelvic cavity, is somewhat constricted off from the main portion above; and this probably represents the dilated sinus.

On the assumption that the atresia of the bladder is primary in origin, it will of course follow that in the male the formation of the penile urethra has proceeded typically, notwithstanding the fact that no communication has been established between it and the urogenital sinus.

As a parallel circumstance, it might be mentioned that in cases of imperforate pharynx the palatine septum and other parts anterior to the œsophagus are normally developed, although no continuity exists between the pharynx and the adjacent portion of the alimentary canal.

Passing from these anatomical considerations, the present specimen in any case obviously demonstrates the importance in amount of the urinary secretion that occurs during intra-uterine life. But if one could be quite sure that the urethra was imperforate from the first, the capacity of the distended bladder and ureters would not certainly give the fluid measure of the actual normal amount of urinary secretion; the cystic disease of the kidneys has to be taken into the calculation.

[1] An account of this specimen is given by Dr. T. W. King, in the 'Guy's Hospital Reports,' ser. i, vol. ii. A paper by Dr. Robert Lee is there referred to ('Med.-Chir. Trans.,' vol. xix) in which one or two other cases of urinary obstruction in the fœtus are culled from different observers.

The fœtus is in other respects perfectly developed, and the cause of death, it may be conjectured, was either the interference with the heart's action occasioned by this enormous abdominal distension, or, for aught that could be proved to the contrary, the fœtus may have died from uræmic coma.[1]

It is generally held at present that the liquor amnii is at first a special secretion of the skin of the fœtus and of the capillaries of the superficial, subamniotic layer of the placenta, but that in the later months of pregnancy it is augmented by the voidance of the fœtal urine, the other sources of the fluid failing.

It has been urged, on *a priori* grounds, that no tissue waste arises *in utero*, and therefore that there is no occasion for urinary secretion; but the movements of the limbs during fœtal life, after the sixth month, are not inconsiderable, whilst there is one muscle the intermittent contractions of which never cease, viz. the heart.

Again, it is not uncommon to find uric acid or urates in the kidneys of infants who have died within a short while after birth. In the last case in which I observed this, the child (which was born in the breech presentation) died on the fifth day; artificial inflation of the lungs was resorted to at the time of birth, but the child never cried properly.

One of the alleged causes of uric acid deposit was here markedly present, viz. deficient oxygenation of the blood. Casper, in his 'Forensic Medicine,' discussing the earlier statements made in regard to the value of this deposit as a positive sign that a child has breathed, states that many observers have recorded similar appearances in children who had died previously to or in birth, and concludes that "the appearances of uric acid deposit in the kidneys of newborn children can no longer be acknowledged to be of any diagnostic value in relation to the question of respiratory life."

[1] That intra-uterine development and growth, however, may proceed independently of renal function, is shown by certain monsters in which both kidneys may be absent. Absence of kidneys occurs, for example, in some sireniform fœtuses. I have lately examined two such of near the full time. There was in each a urinary bladder but no trace of ureters or kidneys. The bladder in one was very moderately distended with mucus, a result comparable to the filling of the gall-bladder with mucus in some cases of complete obstruction of its proper duct. The products of tissue-waste are probably, under such circumstances, eliminated through the placental circulation and ultimately, by means of the maternal blood, through the urinary system of the mother.

In two carefully recorded cases quoted from Schwartz, the children (delivered by forceps) were born with feeble pulsation of the heart, but could not be brought to respire.

Possibly such deposits in the kidneys mark in all cases an intra- or extra-uterine death, accompanied with deficient oxygenation. The occasional presence of such deposits, however, may be cited among the proofs of the renal function in the fœtus, the other and larger proofs of which are the detectability of urea in the liquor amnii, and the result, especially, that ensues in cases of obstruction to the voidence of the fœtal urine, such as the present.

Lastly, in regard to the cystic direase of the kidneys, what relation does this bear to the urethral obstruction? It is natural at first to conclude that the general cystic disease of the kidneys is due to the obstruction in the urinary track below, which obstruction has told back upon the renal pelvis and thence upon the tubuli.

Against such a hasty deduction, however, must be weighed the facts, firstly, that this general cystic disease has often been observed in the fœtus quite independently of any obstruction in the urinary track; and, secondly, that in cases of acquired obstruction in after-life, although the distension of the ureters and pelvis may reach the extreme degree, this general cystic disease never arises. It might be said that, although obstruction in the adult produces hydronephrosis alone, without general cystic disease, it may be different in the fœtus whilst the kidneys are still in process of growth. This argument is easily refuted, for there are specimens of urinary obstruction in the fœtus in which, without any cystic disease, the kidneys are in the extremest condition of hydronephrosis. There is, for instance, a specimen in Guy's Hospital Museum of great distension of the bladder with hydronephrosis in a female fœtus, arising from some not very clear source of obstruction beyond the bladder (2551[4]). Microscopic sections of the kidneys (which I was enabled to make through Dr. Goodhart's kindness) show a layer of renal substance 1·5 mm. in thickness, bounding the sac of the greatly dilated pelvis, but there is no cyst formation whatever in any portion of the tissue, and there is no noticeable fibrosis.

The pathology of the cystic condition in the specimen I exhibit must therefore, I think, be for the present left obscure.

February 7th, 1888.

22. *Stone from a vesical diverticulum.* (*Card specimen.*)

By Thomas Vincent Jackson.

This uric acid calculus, weighing 51 gr., was successfully removed by supra-pubic lithotomy from a diverticulum of the bladder of a man aged 51. The diverticulum was situated on the left side of the base of the bladder at its junction with the posterior wall. Previous unsuccessful attempts to remove the stone had been made by lithotrity, by perineal median lithotomy, and by perineal lateral lithotomy.

Woodcut 7.

On the performance of the two latter operations, the stone was perceived to be beyond the reach of the forefinger, and although its free end was struck with the forceps yet it could not be held by them, and it was found impossible by every effort to dislodge it.

The man made an excellent recovery. *May 15th*, 1888.

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23. *Stone in retroprostatic pouch.* (*Card specimen.*)

By W. Bruce Clarke.

W. M., aged 55, first came to St. Bartholomew's Hospital in 1885 with symptoms of stone and stricture. There was considerable difficulty experienced in introducing a catheter owing apparently to the presence of a calculus in the urethra. Median urethrotomy was performed, a stricture divided, and a stone

removed from the urethra. A sound was then introduced into his bladder, and a second stone detected. Lateral lithotomy was accordingly performed, and a stone over an inch in its largest diameter removed. The prostate was much enlarged.

In 1886, as nearly as possible a year later, a second stone was removed by lateral lithotomy. In July, 1887, he returned with an attack of cystitis and symptoms of another stone, but none was detected on sounding. As his symptoms grew worse, an external urethrotomy was performed, with a view of draining the bladder. There was a considerable amount of phosphatic deposit in the urethra. Owing to the size of the prostate, it was impossible thoroughly to examine the bladder with the finger.

He became gradually worse and died on October 16th, 1887. When the bladder was examined, the cause of the cystitis was revealed: a stone was found about the size of a walnut lying in a pouch behind the prostate. It was so overlapped by the prostate during life that it quite escaped detection. The bladder was largely coated with phosphates. *February 21st*, 1888.

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24. *Bladder from case of prostatectomy and supra-pubic lithotomy.* (*Card specimen.*)

By William Bull, M.B.

Specimen shows a bladder with considerable enlargement of the lateral lobes of the prostate. The most prominent part of the left lobe has been cut away, and an unhealed ulcer the size of a half-a-crown remains. Above this a concavo-convex stone is seen. The stone is now in the situation in which it was during life, and a fair estimate can be made from the concavity of the calculus of the size of the piece of prostate removed.

The patient was a carpenter, aged 71, who had frequently been an in-patient at St. George's Hospital with retention, due to enlarged prostate, which was relieved by catheter. Cystitis supervened, and it was decided to drain the bladder by the perineum. Perineal cystotomy was performed, but from the depth of perineum it was impossible to reach the bladder with the finger. A tube

was introduced, but owing to the irritation it produced could only be kept in for three days. Subsequently the man left the hospital and returned in a few weeks in great pain; urine had to be drawn off every quarter of an hour, and it was decided to drain bladder above the pubes.

Supra-pubic cystotomy was performed in December, 1886, and bladder opened. A calculus was found and removed. This lay above and behind a greatly enlarged left lateral lobe. After removal of stone, the lobe, which formed a prominent tumour, was cut away with scissors by Mr. Holmes. There was free hæmorrhage. The man recovered from the shock of the operation but showed no reparative power, and gradually sank three weeks afterwards. There was some sloughing of the cellular tissue of the pelvis.

At the *post-mortem* extensive granular degeneration of both kidneys was found. *December 6th*, 1887.

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25. *Cancer of the prostate; removal of part of the middle lobe by prostatectomy.* (*Card specimen.*)

By C. Stonham.

The bladder is small and contracted and the walls are thickened. The mucous membrane is corrugated, and at the base it is irregular, owing to the presence of nodules of new growth. The prostate is much enlarged, projecting backwards, and occupying, in the recent state, all the space between the bladder and rectum, the lumen of which was considerably narrowed. The bulb of the urethra is a mass of new growth. Microscopically the tumour is composed of fibrous tissue enclosing large alveolar spaces closely packed with spheroidal epithelium cells. There were numerous secondary deposits in the liver.

The small separate piece is the portion of the prostate removed by operation; a piece of glass has been passed through a false passage in it.

The patient was a man, aged 57, under the care of Mr. C. Heath at University College Hospital. One year before admission there was frequency of micturition attended by pain and difficulty; this

was worse at night. The symptoms increased, and the pain became so intolerable that he was unable to walk on account of it. He had been taught to use the catheter.

On admission, there was a yellowish-white urethral discharge. Mr. Heath passed a No. 13, and drew off the urine mixed with blood. The prostate was enlarged.

On March 14th, 1888, Mr. C. Heath performed prostatectomy, removing the small piece exhibited with curved scissors. The wound in the bladder was partially closed by silk sutures, and a drainage-tube inserted. No urine passed by the urethra until March 27th, and then only about an ounce. The pain remained very severe, necessitating the use of opiates. On April 2nd he walked about the ward and passed most urine by the urethra. He gradually sank and died on April 23rd. The wound had never completely healed. (University College Museum, No. 1808A.)

May 15th, 1888.

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26. *A case of carcinoma of the prostate.* (*Card specimen.*)

By E. Hurry Fenwick.

This bladder was removed from the body of a man, aged 56, who had been an out-patient under my care at the London Hospital. When I first saw him, some six months before his death, he exhibited all the usual symptoms of an "enlarged prostate," and unless I had made a rectal examination I should have been greatly deceived as to the real nature of the case. The right lobe of the prostate was found to be larger than the left. It was "hard as a stone," and irregular in its outline. There was also an ill-defined thickening stretching along the course of the right vesicula seminalis, an offshoot from which could be traced directly across the bladder base to the left vesicula. I made a clay model of the parts[1] for comparison with the same after death, in case I should be able to secure an autopsy. He gradually sank.

Autopsy.—Innumerable secondary deposits of carcinoma were

[1] Author, "Clay Modelling of the Living Prostate as a means of obtaining a Permanent Record of Progressive Disease," 'British Med. Journal,' April, 1887.

found on the surface of the lungs; all were small, some were puckered as if composed of a contracting substance. The right kidney was surrounded by fat. Its capsule stripped easily, its substance was healthy, though rather congested. Its ureter was not dilated. The left kidney presented a remarkable contrast. It was much atrophied by backward pressure, and its ureter was correspondingly dilated. The bladder was well surrounded by fat, but the prolongations of the same along the ureters and vasa deferentia were thick, tough, and adherent to that viscus. On slitting open the bladder the muscular wall was found to be thin, and the mucous membrane smooth, but darkly pigmented. It had evidently been recently congested at the neck. No trace of ulceration existed, not even over the growth to be immediately described. The trigone was covered by six small, flattened deposits of carcinoma, yellow in colour, hard in consistence, and of a clear, translucent aspect. They ranged in size from a pea to a large orange pip, and were evidently situated in the submucous tissue. The orifice of the right ureter had posteriorly to it a similar deposit. The median or "third lobe" of the prostate had been transformed into a small carcinomatous mass, which projected into the prostatic urethra so as to partially block the canal. The prostate was carcinomatous throughout, both lobes being equally affected, its section affording the cupped, unripe-pear appearance of the so-called scirrhous cancer. There were many small, enlarged glands at the back of the bladder, and the left vesicula seminalis was completely transformed into growth. The left ureter was ringed or throttled by cancer, which explained the condition of the corresponding kidney. The internal iliac artery was very atheromatous, the veins were healthy. The entire left lumbar chain of glands was implicated, but that on the right side was free. The denseness of the growth, and the few symptoms afforded are salient features in the case. The disease had evidently commenced in the right lobe of the prostate, had spread to the left, and at the time of death was gradually invading the trigone. But little change beyond the invasion of the left lobe had taken place in the configuration of the base of the bladder since the clay model was taken. *April* 13*th*, 1888.

27. *Tubercular disease of the left vesicula seminalis and left half of the prostate, with extension into the left vas deferens.* (*Card specimen.*)

By C. Stonham.

The prostate is enlarged, and the left lobe infiltrated with caseous tubercles. The rest of the gland is healthy. The left vesicula seminalis is much enlarged, and the section shows the convoluted tubes much dilated, the walls thickened and yellow, and in some places the channels blocked with caseous material.

The abdominal portion of the left vas deferens is thickened. It has been laid open, and the mucous membrane is seen to be affected with tubercle, being pitted with small points of ulceration. The disease ceases abruptly, the lower part of the vas and the epididymis being healthy. The right vesicula and vas are healthy.

The bladder is small, and its walls are thickened; the mucous membrane is healthy except close to the neck, where there are numerous small tubercles, most of which are yellow in colour. The condition was not suspected during life.

The patient, a man aged 26, died from the effects of tubercular disease of the lungs and larynx. The throat had caused him considerable trouble for about eighteen months. He had had syphilis two and a half years before his death. (University College Museum, No. 1904a.) *May 15th*, 1888.

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28. *Cystic tumour of organ of Giraldes.* (*Card specimen.*)

By F. S. Eve.

A circumscribed tumour composed of a number of cysts with thick fibrous walls. The cysts all contained clear fluid except one, in which was a milky fluid rich in chlolesterine. The tumour was removed from a boy aged 7. It lay near the testicle, and was diagnosed as a cystic tumour of that structure; but on dissection it was found to be "clear of both the testicle and cord."

It probably originated from the tubular Wolffian remains, known as the organ of Giraldes, and is the only completely satisfactory specimen of the kind I am aware of. (Presented to the Royal College of Surgeons by Mr. H. Mallins.) *April 4th*, 1888.

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29. *Cystic disease of the testis.*

By A. Quarry Silcock, M.D.

The specimen shown was obtained from the body of a middle aged man who died in one of the medical wards of St. Mary's Hospital. The scrotal swelling to which it gave rise was thought

Woodcut 8.

Cystic disease of testis (1 inch obj.). Drawn by R. Ostlere, M.B.

at the autopsy to be an ordinary vaginal hydrocele, but on being cut into subsequently, after having been put aside for some time in spirit, the following condition of things was apparent. The body

of the testis was the seat of an interstitial fibroid overgrowth, seemingly radiating from the mediastinum testes, with an accompanying progressive cystic dilatation of the seminiferous tubules. The cysts, to the naked eye, varied in size from a pin's head to a pigeon's egg, but microscopically every stage between a well-formed seminiferous tubule and the fully-developed cyst was observable. A scrotal tumour was thus formed, which consisted almost wholly of tough-walled cysts, some of these having ruptured, owing no doubt, to absorption by pressure of the intermediate fibrous tissue, so giving rise to multilocular cystic cavities of considerable size. The contents of the cysts varied, being a clear hydrocele-like fluid, in the larger, or as in the smaller, consisting of a thick, glairy, more or less opaque fluid, or of caseous material.

The disease appears to be analogous to some cases of cystic disease of the breast with general dilatation of the ducts, and to pure cystic disease of the kidney. There is no evidence to the naked eye, or microscopically, that it is in any way connected with the formation of a neoplasm, such as an adenoma or fibroma.

The vas deferens below the external ring was permeable and healthy, but whether this was the case higher up is uncertain, since the cord above that point was not examined. *May* 1*st*, 1888.

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30. *Very early malignant disease of the testis.* (*Card specimen.*)

By William Henry Battle.

Testis, the seat of new growth, removed from a boy aged $3\frac{1}{2}$, on October 22nd, 1886. He was said to have fallen from a cart some little time previous to the time that the tumour was noticed, but was not known to have injured the testis. There had been swelling of the testis for three months, with more marked increase for six weeks.

There was no evidence or history of constitutional disease. The boy was healthy, excepting for a painless swelling of the right testis. This was not larger than a filbert nut, but was nearly three

times the size of its fellow. Quite painless, and not tender. The tumour did not involve the whole organ, only the part near the epididymis, where it was hard and nodulated, almost craggy; the cord was free. No evidence of disease elsewhere.

After removal, the body of the testis was the seat of the enlargement (it had simulated disease of the epididymis, and was diagnosed by some as tubercular disease of that body). There were enlarged veins on the surface. No fluid in the tunica vaginalis. On section it was yellowish, hard; surface convex and not mottled.

Mr. Shattock, who carefully examined the growth microscopically, pronounces it to be a myxo-sarcoma ("cylindroma"). "It is a kind of cylindroma,—branching columns of connective-tissue cells in a lax mucous connective tissue."

The patient left the hospital on November 22nd, 1886, the wound being quite healed, and his mother writes me that he continues in the best of health, and without any further evidence of local disease. *May 15th*, 1888.

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31. *Papilloma of both Fallopian tubes and ovaries.*

By Alban Doran.

[With Plate XII.]

Mrs. R—, aged 31, shopkeeper, was admitted into the Samaritan Free Hospital in December, 1887, on account of a painful hypogastric tumour.

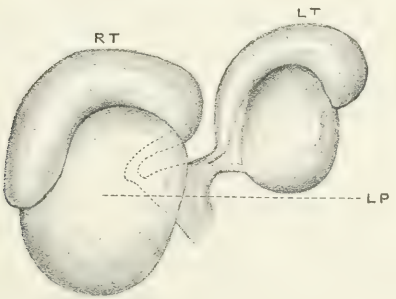
The catamenia appeared when she was fourteen; she was married at the age of nineteen. For four years after that date she continued in good health. The period was regular and copious, as before marriage, but she did not become pregnant. Seven years ago, when she was twenty-four, she was attacked with severe pelvic inflammation, and remained for nine weeks in the Royal Free Hospital under the care of Dr. Hayes, Physician for the Diseases of Women. A tumour was then noticed in the hypogastrium; the patient declared that it slowly disappeared. Since that illness the

catamenia had been scanty, painful, and of only two days' duration. The patient did not become pregnant.

For twelve months before admission the patient was troubled with lumbar pains, abdominal swelling, and dysuria during the period, which persisted, it may here be remarked, notwithstanding the total disorganisation of both appendages and the absence of any signs of hæmorrhage in the fluid in the tubal cysts. Three months before admission, when she was under the care of Dr. Grigg, the tumour reappeared and slowly enlarged. The patient became unfit for business, and begged that it might be removed.

The patient was fairly nourished. The abdomen was not distended above, and there was resonance in the flanks. The hypogastrium was prominent, owing to the presence of a tumour which did not reach quite as high as the umbilicus, and extended downwards into the pelvis, pushing the cervix uteri against the left side of the pelvic cavity. Another tumour, much smaller, lay in the hypogastrium to the left of the middle line, hardly reaching the pubes; it was very freely movable, and felt like a pedunculated subperitoneal fibroid. Both tumours felt elastic, like soft fibroids, fluctuation being, at least, very obscure. Diagnosis was extremely uncertain. An exploratory operation was decided upon. It was performed by Dr. Bantock on December 21st, 1887. As I assisted the operator, I had the advantage of being able to note the extremely complicated relations of the tumours which were discovered.

Woodcut 9.



When the abdominal cavity was laid open the smaller tumour, which lay to the left, looked like a coil of small intestine adherent to a cyst. It was tapped, and clear, glairy, apparently ovarian

fluid escaped. The coil proved to be the left Fallopian tube, dilated. The cyst below it was drawn with great difficulty out of the abdomen, for it burrowed between the layers of the broad ligament, and had to be enucleated inferiorly. The lower part of this cyst lay close to the side of the body of the uterus, which was above the pubes, and covered in front by the larger tumour. The sketch (Woodcut 9) shows these relations, as detected in the course of the operation (L.P. = level of pubes). The larger tumour lay more in front of the uterus, but I have drawn it as though pushed somewhat outwards in order to display the connections of the left tumour. A pedicle, consisting of the uterine end of the left tube, part of the broad ligament, and a stout band (no doubt the ovarian ligament) running to the cyst, was formed by transfixion close to the uterus. The left tumour was then cut away.

The right tumour proved to be a similar but larger growth, and was treated in a similar manner. It burrowed very deeply into the pelvis, and its removal was exceedingly difficult; its pedicle was transfixed close to the right extremity of the fundus uteri. A drainage-tube was inserted into Douglas's pouch. The patient made a rapid recovery, the temperature never exceeded 99·6°, and she was discharged in good health on January 11th, 1888.

Before describing the tumours (R. C. S. Mus., Nos. 4584 B and C) I may note that the abdominal viscera exposed during the operation appeared healthy and free from inflammatory or neoplastic deposits, and that the peritoneal cavity contained no free fluid.

The tumours are represented in Plate XII, figs. 1 and 2. In describing them I am unable to give very precise measurements. They were very large as they lay in the body distended with fluid, but shrank considerably after removal and preservation in spirit, though I have counteracted the shrinking, to a certain extent, by stuffing the cystic cavities with wool before immersing the specimens in dilute methylated alcohol.

The right tumour weighed $7\frac{1}{2}$ oz. when fresh, and contained over a pint of fluid. As now preserved the uterine half of the tube (fig. 1, *a*, *b*) is stretched and tortuous, its walls are very thick, and its mucous membrane bears small papillomata, which were more conspicuous and highly vascular when the specimen was fresh. This part of the tube is invested by the broad ligament much thickened by old inflammation. The outer half of the tube is dilated, forming an oval cyst measuring in its present half-collapsed

DESCRIPTION OF PLATE XII.

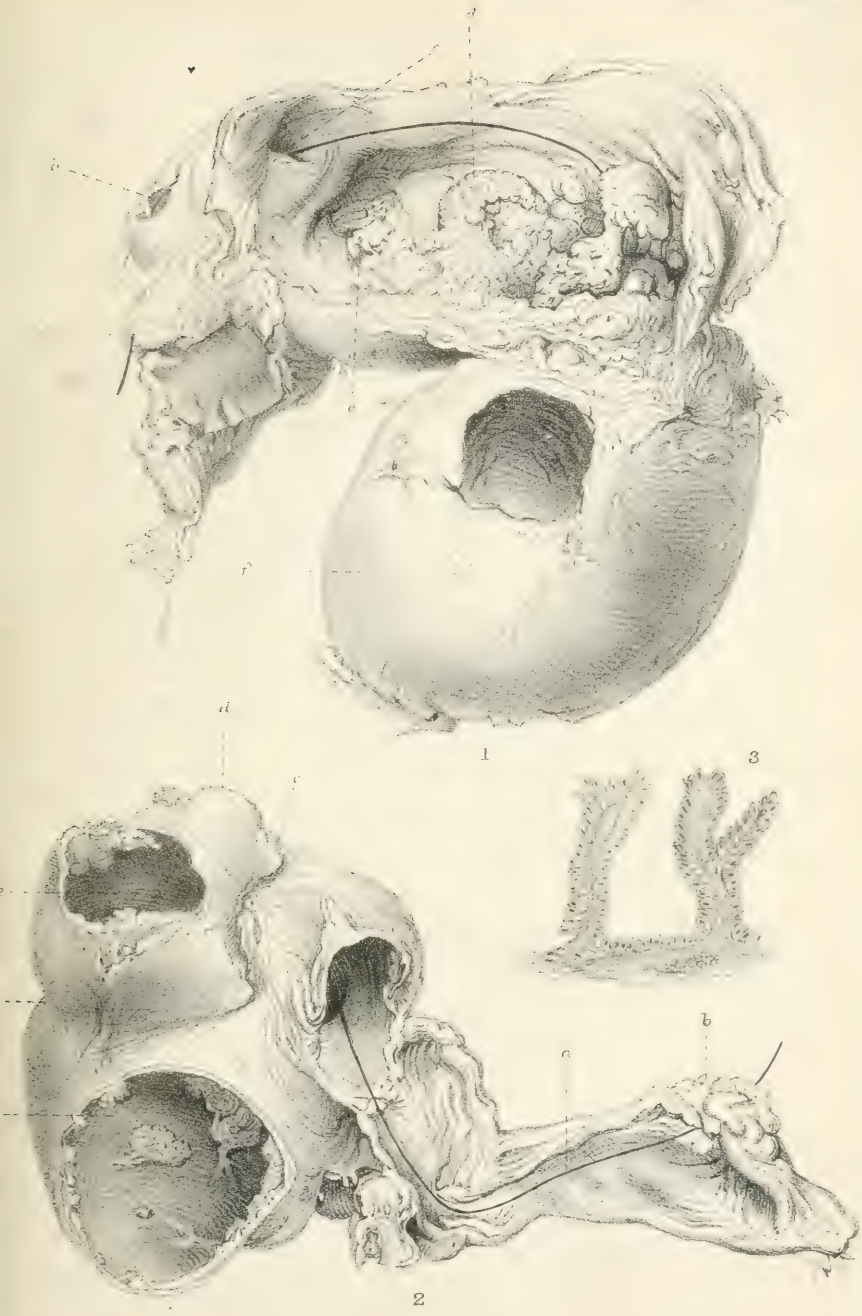
To illustrate Mr. Alban Doran's case of Papilloma of both Fallopian Tubes and Ovaries. (Page 200.)

From drawings by Mr. H. R. W. Lewin.

FIG. 1.—Right tumour, seen from behind. *a*. Cut end of the Fallopian tube, which was divided close to the right extremity of the fundus uteri. *b*. Inner half of the tube laid open, showing its thickened walls; it winds along the back of the specimen (as here placed), and its cavity, laid open, is only visible to a limited extent in this drawing. Below it lie the thickened layers of the broad ligament. *c*. Valvular opening between *b* and the dilated part of the tube, which is completely exposed, showing (*d*) masses of papillomata, and (*e*) a cavity in its thickened walls also bearing papillomatous growths. A bristle has been passed through *a*, *b*, and *c*, and rests against a papillomatous mass. *f*. The right ovary, forming a unilocular cyst which does not communicate with the tube. The small papillomatous growths on its inner wall are not indicated. ($\frac{2}{3}$ nat. size.)

FIG. 2.—Left tumour, seen from behind. *a*. Uterine half of the Fallopian tube laid open from *b* (where it was cut through, close to the left extremity of the fundus uteri) up to its dilated portion, and traversed by a bristle. *c*, *d*, *e*. Outer portion of tube curving forwards (at *c*), then upwards (at *d*), then backwards (at *e*). This portion contained masses of papillomata; some are indicated in the drawing at the upper and lower border of the hole cut in the tube at *e*. *f*. Outer limit of the line of demarcation between the tube and the ovary. *g*. Left ovary dilated into a single cyst bearing papillary growths, some pedunculated. (Nat. size.)

FIG. 3.—Microscopic section of a papillomatous outgrowth from *e*, Fig. 2, as seen under a low power. The papillæ are very fine, like villi, and bear columnar epithelium; one papilla is branched.



West, Newman & Co. lith.

condition over four inches horizontally by about two and a half vertically. It communicates with the uterine half by a crescentic valvular orifice (*c*). A similar condition is seen in Woodcut 9, p. 242, in the thirty-eighth volume of the 'Transactions' (1887), which represents a tubo-ovarian cyst, developed as a result of chronic inflammation. Large papillomatous masses (*d*) grow from the mucous membrane of this dilated portion of the tube. In its extremely thickened walls, a short distance below the valvular orifice, is a cyst, which, when fresh, contained nearly one drachm of yellowish glairy fluid without any pus-corpuscles. Papillomatous outgrowths (*e*) spring from the lining membrane of this cyst, which may have developed from one of the pouches or cavities formed in the "follicular salpingitis" of Martin.[1]

Below the dilated part of the tube lies the ovary (*f*) reduced to a single cyst, almost spherical, and about four inches in diameter. Its lining membrane bears, at certain points, very fine, almost villous papillary elevations. This cystic ovary does not communicate with the dilated part of the tube, to which, however, it is intimately adherent. At the area of adhesion I detected signs of atrophy of the cyst wall, and a distinct valvular elevation appeared to mark the breaking-down either of a secondary cyst or of the main wall, as so often seen in large multilocular ovarian cysts. This appearance, which is not clearly displayed in the specimen as permanently mounted, indicates the first stage in the formation of a true tubo-ovarian cyst as a result of chronic inflammation of the appendages (see Woodcut 9, 'Transactions,' vol. xxxviii, above referred to). From what I have observed (ibid., p. 243), dilated tubes and ovaries which have become cystic through inflammatory changes, may, I conclude, fuse together to form tubo-ovarian cysts. Thus a tubo-ovarian cyst arising in this manner represents an atrophic process following inflammation; this theory will be further considered. I admit that Dr. Griffith has noted that, according to minute descriptions of specimens of tubo-ovarian cyst already

[1] "Ueber Tubenerkrankung," 'Zeitschrift für Geburtshülfe,' vol. xiii, part 2. The "follicular depressions" of Martin may be produced by the union of the edges of secondary plicæ through inflammatory adhesion. I have seen this condition in a section prepared by Dr. Griffith from a tube removed during life. In the present case, however, the cyst is separated from the main cavity of the tube by part of the thickened tubal wall, and therefore must have been otherwise developed.

published, no mention is made of any appearances which show that the tubal and cystic cavities have united in this manner.[1] In this particular case I am speaking solely of tubo-ovarian cysts developed as a result of chronic inflammation.

This cystic ovary burrowed deeply into the layers of the pelvic peritoneum, whence it was enucleated during the operation. All trace of ovarian tissue has disappeared, as is the rule early in the development of papillomatous cystic disease of the ovary.[2]

The left tumour (fig. 2) weighed 3 oz. when fresh, and contained a quarter of a pint of fluid. As here preserved the uterine half of the tube (*a*, *b*) measures about three inches in length. It dilates suddenly and has been laid open since the operation posteriorly as far as the dilatation. The mucous membrane, intensely vascular when recent, bears no papillary growths; the muscular and serous coats are much thickened. The outer half of the tube, greatly distended with fluid before removal by operation, bends forwards from the dilated part of the inner half at *c*, and then curves upwards and backwards (*d*, *e*), becoming yet more dilated, and ending on the surface of the ovary. The line of demarcation at *f* is still perceptible, but was more strongly marked in the recent state. All the outer half of the tube bears masses of papilloma. The ovary is dilated into a single cyst (*g*) hardly two inches in diameter, but originally much larger. Its lining membrane bears papillomata more developed than in the cystic ovary on the other side, some forming polypoid growths springing from short pedicles. This ovarian cyst does not communicate with the dilated tube. It was partly shelled out of the pelvic peritoneum during the operation, and some of the serous membrane still invests the cyst at the back of the specimen as now mounted. This piece of peritoneum removed with the cyst was so thickened that I took it at first for normal ovarian tissue, but found no such tissue on careful search. The ovarian ligament formed a short stout cord when the specimen was fresh, but it has shrunk up in process of preparation, and lies

[1] "Tubo-ovarian Cysts," 'Trans. Obstet. Soc.,' vol. xxix. For a description of tubo-ovarian cysts in London museums see my observations in the discussion on Dr. Griffith's paper, ibid., p. 302.

[2] "Papillary Cysts of the Ovary," 'Transactions,' vol. xxxiii, p. 207. Plate IX, fig. 2, represents a cyst bearing fine papillomatous growths resembling those which spring from the wall of the cystic ovary in the present case, though not indicated in the drawing.

on the under and inner part of the cyst, unrecognisable to any person who has not had the opportunity of examining the specimen when fresh. I took care to note minutely the relations of all the structures as displayed during operation, and also after the removal of the specimens whilst they were still fresh. I admit that now they are hard to recognise. The papillomata have shrunk everywhere, and the characteristic appearance caused by their vascularity when fresh has entirely disappeared.

Thus there is papillomatous disease of both tubes and ovaries, but no true tubo-ovarian cyst on either side, although the conditions under which a cyst of that kind is most frequently developed were present (see 'Transactions,' vol. xxxviii, p. 241), and, indeed, in this case had no operation been performed the tubal and ovarian cysts might have ultimately opened into each other.

The papillary growths in the right tumour closely resembled those in the case of papilloma of the Fallopian tube which I described in the 'Transactions,' vol. xxxi, p. 174, but are not so generally diffused over the tubal mucous membrane. In the left tumour they are rather finer even in the part of the tube near *e*, fig. 2, and certainly finer still in the ovary *g*, where, when fresh, they looked and felt like hard, brittle warts. Fig. 3 represents the microscopical appearances of a piece of papilloma from *e*, fig. 2. The papillæ are fine and elongated, villiform in fact; some are branched. They bear a distinct investment of columnar epithelium, but I failed to detect cilia; however, the specimen has certainly suffered from remaining immersed in water for several hours after the operation, and the epithelium shows signs of detachment by maceration in many places. Under these circumstances, cilia, as every microscopist knows, disappear.

I speak of these growths as papillomata because they consist of very vascular papilliform projections from the mucous membrane and from the lining membrane of the ovarian cysts, and bear epithelium after the fashion of normal papillæ. They are identical with the growths which I described in the thirty-first volume of the 'Transactions,' and the growths described by Dr. Coblenz in his important memoir "Zur Genese und Entwickelung von Kystomen im Bereich der inneren Sexualorgane," published in Virchow's 'Archiv,' vol. lxxxiv, 1881, and in his "Das Ovarialpapillom," ibid., vol. lxxxii, 1880. Mr. Bland Sutton and myself have since the appearance of these papers made fresh researches in the same

direction, but to Dr. Coblenz is due the honour of having first clearly demonstrated the pathological and morphological significance of papilloma of the ovary as a disease perfectly distinct from the common multilocular ovarian cyst. The growths on the surface of the tubal mucous membrane resemble the growths in the ovary which the above-named observers, as well as myself, have uniformly described as papilloma, and differ greatly from the adenomatous vegetations which grow within the loculi of many multilocular ovarian cysts. How far tubal and ovarian papilloma have any relation will presently be discussed.

This case is the third where papilloma of the Fallopian tube can be said to have constituted a distinct disease. In all these cases there was evidence of previous pelvic inflammation. In the first case ('Trans. Path. Soc.,' vol. xxxi, and Mus. R. C. S., Path. Series, No. 4584), where the papillomatous growths were exuberant, the ostium of the affected tube was patulous, and ascites was present; the corresponding ovary was not enlarged. In the second ('Trans. Obstet. Soc.,' vol. xxviii) the affected tube was closed and there was no ascites, the growths were exuberant, the ovary was embedded in adhesions, and was apparently free from papillomatous disease. In the third case, above described, the disease affected both tubes, which were closed, and there was no ascites, the growths were less exuberant; both ovaries were papillomatous. The first case is yet living, though the late Professor Schröder, who referred to it,[1] believed that the papilloma was evidently malignant. The second died on the fourth day after operation. The disease certainly did not appear to be malignant either in this or in the third case. The significance of ascites I have already discussed at length ('Trans. Obstet. Soc.,' vol. xxviii); in the case here described it was absent because, or at least chiefly because, neither tubes were patent towards the ostium.

The relation of papilloma of the ovary to papilloma of the tube may next be considered. There is no directly pathological relation between the two diseases. Papilloma of the ovary apparently arises from relics of the parovarium which are found in the hilum of the ovary (paroöphoron) or even stray into the ovarian parenchyma proper among the follicles. I have demonstrated this theory in papers which are published in the thirty-second, thirty-

[1] 'Die Krankheiten der weiblichen Geschlechtsorgane,' 7th edition, 1886, p. 361.

third, and thirty-fourth volumes of the 'Transactions.' No such relics enter into the tissues of the Fallopian tube, where papillomatous disease appears more evidently as a result of inflammation. Yet although papilloma of the ovary is more essentially a tumour in its clinical characters (however it may differ from the common multilocular cyst pathologically), there is evidence that it may follow old pelvic inflammation. There was "uncontrollable menorrhagia," and other symptoms of what is generally termed "chronic disease of the appendages," in one of the cases which I have described in the 'Transactions.' In the case which forms the subject of this paper, the history of pelvic inflammation was very clear.

Turning to the condition of the tubes in this case, there can be little doubt that it was due to the pelvic inflammation. Mr. Sutton has frequently detected a slightly papillomatous condition of the tube caused, he believes, by the extension of gonorrhœa from the lower part of the genital tract. Dr. Bumm, I may here remark, claims to have discovered that the pathology of "unmixed" gonorrhœa may best be studied in the tube rather than in the vulva or the vagina, since the gonococci readily reach the tube, whilst the pyogenic streptococci, which so often enter gonorrhœal discharges in the vagina ("mixed" or "compound infection"), seldom pass upwards into the higher portions of the genital tract.[1] Mr. Sutton has no doubt "that the growths of the tube are in nature warts, similar in principle to those found in other situations, namely, overgrown papillæ, the result of continued irritation." In the case of the tube the papillomata are "no doubt . . . . simply enlarged and overgrown rugæ characteristic of its mucous membrane" ('Trans. Obstet. Soc.,' vol. xxviii, p. 247). I have already referred to Dr. Martin's "follicular salpingitis."

From what has been said in the two preceding paragraphs I infer that there was, in this case, a more or less simultaneous papillomatous degeneration of the tubes and ovaries due to a common cause, the attack of severe pelvic inflammation seven years previous to the operation. Ever since the inflammatory attack the patient suffered more or less, the catamenial functions being permanently deranged. Whether the attack represented gonorrhœal infection or not must remain obscure. The papilloma of the tubes and

[1] "Ueber gonorrhoische Mischinfectionen beim Weibe," 'Deutsche med. Wochenschrift,' December 8th, 1887.

ovaries represent, I believe, an essentially atrophic condition, like the tubo-ovarian cyst which I described in the thirty-eighth volume of the 'Transactions' as the result of chronic inflammation. In the present case the atrophic cystic changes were also proceeding in the direction of the development of tubo-ovarian cysts, especially on the right side, as I have already indicated, though I do not wish to be too positive on that point.

I do not think that the disease could have been congenital. Dr. Griffith, in the monograph already quoted, gives reasons for supposing that some forms of tubo-ovarian cysts are congenital. In this case there is no tubo-ovarian cyst on either side, though the condition appears to be developing on both. The patient menstruated regularly before the attack of pelvic inflammation, and the previous sterility may be explained by many more probable theories than the supposition of congenital malformation.

February 21*st*, 1888.

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32. *Primary cancer of the Fallopian tube.*

By Alban Doran.

[With Plates XIII and XIV.]

Mrs. B—, aged 48, was admitted into the Samaritan Free Hospital, under the care of Mr. J. Knowsley Thornton, in February, 1888. Mr. Malcolm prepared the following notes of the case :—"Married twenty-three years (family history unimportant), one child born twenty-two years ago, no confinement since. Admitted with a tender, hard, mobile tumour in the right groin, two inches in diameter, reaching to level of anterior superior iliac spine. *Pelvic examination.*—Cervix small, feels healthy, lies far back, points backwards. Tumour of groin is very closely connected with right cornu of uterus. The period had previously been always regular, little pain, much show. Twelve months ago it began to diminish. Last period August, 1887."

For three years the patient had been subject to a watery, inoffensive, and occasionally sanious vaginal discharge, which often

contained small pieces of membranous material. For this the patient consulted Dr. Amand Routh in October, 1886. In January, 1887, he dilated the cervix, introduced a curette, and scraped away about two drachms of a red fungous growth from the fundus. At that time no pelvic tumour could be detected. A fortnight later an attack of pelvic inflammation occurred, with swelling of the left leg and left labium majus. This complication may, as Mr. Thornton suggested, have been caused by irritation of the malignant growth, already developing though too small for detection. Every surgeon knows that malignant tumours are very intolerant of operation in their vicinity. As the symptoms and pain subsided, a tumour was discovered, rising to the right above the pubes. The tumour grew slowly and the patient lost flesh.

On March 1st, 1888, Mr. Thornton operated. The abdominal walls were fat, and bled freely on section; there was no free fluid in the peritoneal cavity. The course of the operation is thus described by the operator:—"Tube exposed, much distended, soft and elastic, punctured with fine trocar, then with aspirator. Only a little ill-smelling bloody serum escaped, with fragments like those coming from the vagina. Punctures closed by square-headed forceps and corrosive sublimate lotion freely used. Large forceps put on to the tube close to the uterus. Then the outer part of the tube, which had curled inwards over the ovary and behind the uterus, was gradually separated from both, the adhesions being very fine, so that I could only tear away part of the ovary, leaving the rest sticking to the broad ligament. When the tube was sufficiently freed, I transfixed and tied in two halves, but on cutting away was obliged to put on several pairs of forceps, as vessels spouted. I then put another ligature round the whole, transferring large forceps to distal side before tying ligatures. The end of the tube left in the stump was patent, but seemed fairly healthy; one small portion of the diseased tissue was removed on the cotton-wool dipped in corrosive sublimate lotion used for cleaning it out." (The "diseased tissue" was probably decolourised clot, with which the uterine end of the removed portion was stuffed.)

"I then with much difficulty removed the left tube and part of a small cystic ovary, the adhesions being much denser than on the right side, and some separate ligatures being required for adhesions deep in the pelvis. The uterus was not so large as I thought

at first, and seemed fairly firm and healthy, so that I rather doubted if there was any of the growth in it." A glass drainage-tube was introduced with some difficulty, as the pelvis was blocked with firmly adherent intestine. The patient made a good recovery, the temperature never rising above 101·2°; the tube was removed on March 4th. She was sent back to the convalescent ward on March 7th, and discharged at the end of the third week of the same month. [In August, 1888, she was in good health.]

The mucous membrane of the left tube was thickened, but free from any morbid growth, as was likewise the ovary. The right tube was of the characteristic legume form observed in most tubes when dilated beyond a certain degree, but there were traces of two convolutions on its inner third. The tube, when collapsed, measured about five inches in length.[1] I cut away a large piece of the anterior wall immediately after the operation, and took it at once to the Physiological Laboratory, St. Bartholomew's Hospital, where Dr. Vincent Harris kindly prepared for me some sections. A reddish fluid, with broken-down particles of solid matter, escaped from the tube as it was opened. Almost the entire surface of the interior of the tube was covered with soft and highly villous growth, of a bright red colour when fresh, no normal mucous membrane being left. Only about one inch and a half (Plate XIII, fig. 1, *b*), nearest the uterine end, remained free from the new growths; here the channel of the tube was firmly packed with partially decolourised clot, and the mucous membrane was thickened and rigid. The tube was completely obstructed at its outer extremity, and no trace of the fimbriæ remained.

The serous coat of the tube was little, if at all, thickened; it did not show clearly on section; thus *e*, fig. 1, is the muscular coat and not the peritoneum. The muscular coat was thick and free from any cavities, like, for example, the sinuses observed in Martin's "follicular salpingitis."[2] It was invaded at certain places (fig. 1, *d*) by the growth, which nowhere perforated the serous coat. The submucous tissue was very conspicuous when the specimen was fresh; it was thickened to the extent of from one eighth to one quarter of an inch at many points; it has become thinner and quite opaque through the action of spirit. In some

[1] The specimen is now in the Museum of the Royal College of Surgeons, No. 4584D.

[2] "Ueber Tubenerkrankung," 'Zeitschr. f. Geburtsh. u. Gynäk.,' vol. xiii, pt. 2.

DESCRIPTION OF PLATES XIII AND XIV.

To illustrate Mr. Alban Doran's paper on Primary Cancer of the Fallopian Tube. (Page 208.)

From drawings by Mr. H. R. W. Lewin.

Plate XIII.

Fig. 1.—A considerable portion of the anterior part of the wall of the tube has been removed to display the extent of the disease. *a*. Uterine end of the tube divided at the operation. A black bristle has been passed through it and along the channel of the tube. *b*. Portion of the tube near the uterine end, free from growths. *c c c* Masses of cancerous growth springing from the inner surface of the posterior part of the tube. *d d*. New growth invading the muscular coat, *e e*, which is elsewhere more or less free from disease. At *d'* a very solid deposit lies in the submucous tissue. *f*. The ovary converted into a mass of tumour substance. *g*. Cut surface of the broad ligament, which is infiltrated with new growth.

Fig. 2.—Section of cancerous growth invading the anterior part of the wall of the tube, as at *d*, fig. 1. ($\frac{1}{8}$″ obj.)

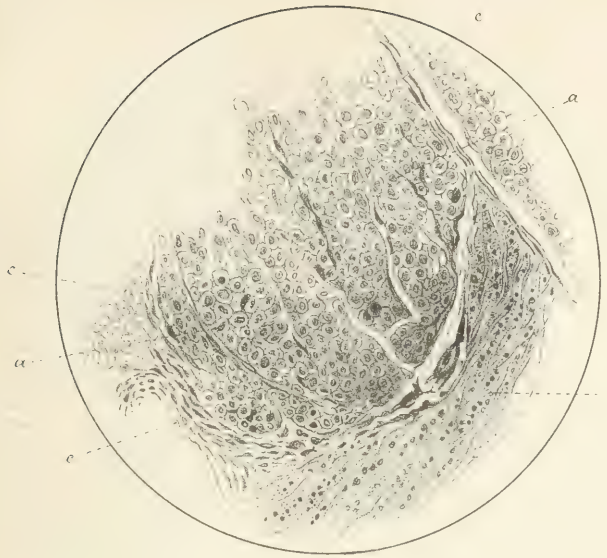
a a. Part of a group of large polymorphous cells. *b*. Part of a trabecula bounding the group of cells showing small-cell infiltration. *c c c*, Muscle-cells indicating remains of the muscular coat of the tube.

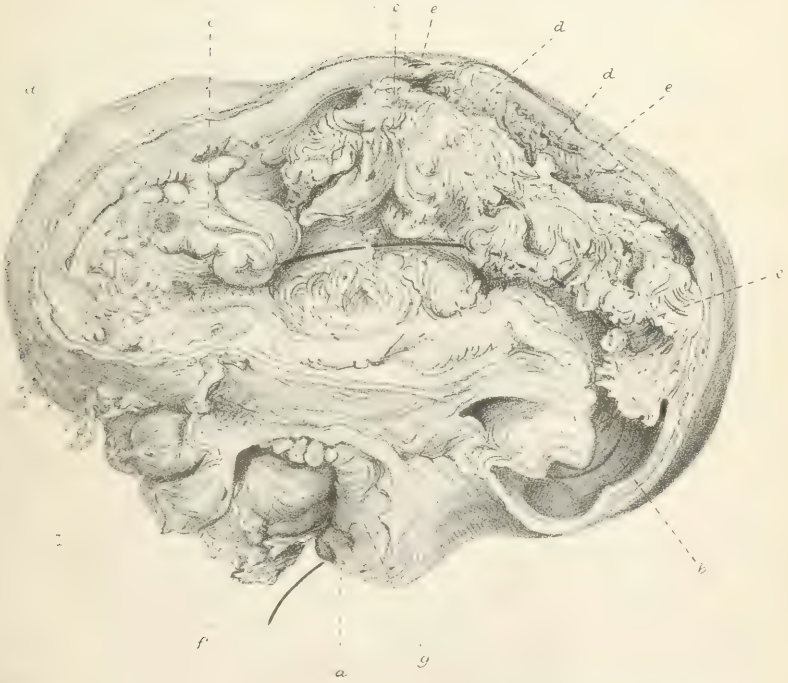
Plate XIV.

Fig. 3.—Tubular structure surrounded by cells, seen in another part of the same section. *a*. The tubule lined with cylindrical ciliated epithelium. *b b*. Large cells surrounding the tubule; they are arranged somewhat spirally and prolonged outwards into the stroma at *c c*. Further on at *d* are larger cells. Some of the cells, *b*, are arranged as though they were inside tubules choked by proliferation of epithelium. ($\frac{1}{8}$″ obj.)

Fig. 4.—Section from the firmest part of the ovary (fig. 1, *f*). *a*. Trabecula infiltrated with small cells. *b b*. Trabecula bearing small spindle-cells. *c c c*. Part of a large collection of epithelial cells showing large spaces (*d d*) formed by vacuolation. The "signet-ring" appearance is indicated in some of the cells. ($\frac{1}{8}$″ obj.)

2.





H.P.W.L. del

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places (*d'*) the submucous tissue was invaded; this of necessity was the case wherever the growth had reached the muscular coat.

The broad ligament was in parts nearly one quarter of an inch in thickness, and infiltrated with the new growth (*g*). It was shortened and crumpled up, as in chronic inflammatory diseases of the uterine appendages. A thin-walled cyst projected from its posterior surface.

The ovary was almost spherical, and measured in its long diameter about one inch and three quarters. It was converted into five or six pale, fleshy lobes, each about one quarter of an inch in diameter, soft and yellowish-white in colour. Between these lobes were recent blood-clots, possibly effused on the tightening of the ligature during operation. No trace of normal ovarian tissue could be detected. A piece of the ovary was preserved, when fresh, for section.

The microscopic appearances of the sections taken from the anterior wall of the tube are shown in Plate XIII, fig. 2. The portion of tube-wall prepared for the microscope was nearly one quarter of an inch thick, and on naked-eye examination it was seen to be partly invaded by the new growth. Throughout the section were wide spaces filled with large cells (*a*). The nuclei of these cells were also of large size. Some were undergoing division; in some the "signet ring" appearance was seen. The fibres of the stroma did not appear to pass between individual cells, nor could I detect any connection between any cell and the stroma. In the immediate neighbourhood of every collection of large cells was a highly alveolar tissue consisting of a network of more or less stout fibres enclosing very elongated spaces, which were stuffed with small round-cells (fig. 2, *b*). In some parts of the sections the network was composed of the muscular fibres of the middle coat of the tube (*c*), and muscle-cells were even to be seen amidst the collections of large cells, as is indicated in fig. 2. At first I thought that the trabecular tissue *b* represented scirrhous cancer, the masses of large cells indicating the softer part of the malignant growth, but after careful comparison I noted that the small round-cells were probably not cancerous at all, but were connective-tissue cells, produced by a process of proliferation observed in the neighbourhood of a cancer in any organ. In this opinion I was confirmed by Mr. Butlin, Mr. Bowlby, Mr. D'Arcy Power, and others who are experienced in general pathology, and Dr. Griffith, who

has prepared a large series of specimens illustrating the progress of uterine cancer, informs me that tissue like *b* in fig. 2 is constantly seen around the collections of epithelial cells in that disease.

There were several other remarkable appearances in the sections. Bundles of muscular fibres, divided transversely, bore a strong resemblance to collections of epithelial cells on superficial examination. Some parts of the sections were highly vascular. The peritoneal surface of each section showed some remarkable minute crypts, which were in some cases bifurcated, and I noted secondary bifurcations in a few of these follicular depressions. The deepest were hardly a fiftieth of an inch in depth. These crypts were remarkable, as they caused the serous surface to be taken at a first glance for the mucous surface, and, moreover, on naked-eye examination the serous coat appeared perfectly smooth. Possibly the depressions were caused by irregular contraction of the subjacent tissue, due to the morbid infiltration. Not a trace of the normal mucous surface could be found in the infected part of the tube, in fact, it had been entirely replaced by the morbid growth, and the microscope proved what was almost as plain to the naked-eye. Deeper down, towards the serous coat, the disease was certainly less advanced. Here I detected two or three well-formed tubules, lined with columnar ciliated epithelium and surrounded by a wide area of large cells (Plate XIV, fig. 3). In this area (*b*) some of the flat cells were arranged as though they represented tubules choked with epithelium. This would indicate that the disease arose from the epithelium of glands connected with the tubal mucous membrane. The cancer-tissue resembled gland-cancer elsewhere rather than epithelioma of a mucous or other free surface.[1] I shall presently have occasion to refer to this subject.

In sections prepared from the ovary I detected the same appearances as in the tube, that is to say, there were extensive collections of large flat cells (Plate XIV, fig. 4, *c*) surrounded by trabeculæ in which the connective-tissue cells were undergoing proliferation (fig. 4, *a*). In other parts I fancied at first that I could trace groups of

[1] In a recently published memoir, "Ueber den primaren Krebs des Ileum," by Dr. Otto Lubarsch ('Virchow's Archiv,' Band cxi, Heft 2, February, 1888, Tafeln viii, ix), the author demonstrates that primary cancer of the ileum arises in Lieberkühn's glands. According to his drawings that local form of cancer closely resembles cancer of the tube in its microscopic characters.

round-cells, each cell being connected by a process to meshes of apparently retiform tissue, which surrounded the entire group. But on further consideration, and examination of the sections with Mr. Eve, I agreed with him that the "small round-cells" were probably large nuclei, the apparently retiform tissue representing vacuolation of protoplasm, such as is seen in cancer. In some of the clusters of epithelial cells this process of vacuolation was not so advanced, but had already produced round or oval holes (fig. 4, *d*, *d*) with more or less regular outlines. In the trabeculæ, although, as already stated, round-cells (fig. 4, *a*) predominated, I found occasional clusters of small spindle-cells (fig. 4, *b*), which possibly represent the spindle-celled tissue of medullary carcinoma figured and described in S. W. Gross's 'Practical Treatise on Tumours of the Mammary Gland,' fig. 23, p. 134 in the 1880 edition.

I have above described what I observed, and I do not think that there can be much doubt that the morbid growth in this case was cancer and not sarcoma.

The clinical and pathological evidence tends to prove that the tube was the primary seat of disease. Considering that there was vaginal discharge for three years, much broken-down material escaping till the date of operation, that the uterus was scraped with a curette, and that fourteen months later it was found free from disease, whilst the tube contained not only a new growth but also fluid and broken-down matter resembling the discharge, that new growth was certainly tubal and not uterine. The ovary was cancerous, but evidence was against its being the original seat of disease. The history of the discharge favoured the view that the tube had been involved for a long time. The growths within the tube were well developed, the tube being consequently much enlarged. On the other hand, the ovary, although entirely converted into a mass of new growth, was still small, as though but recently invaded. Had the tumour detected by Dr. Routh early in 1887 been ovarian, the ovary would have formed a very large mass of malignant disease by March, 1888, for solid malignant ovarian tumours grow very quickly. The tube is ever slow to become invaded in primary cancer or sarcoma of the ovary, and when attacked it is usually the serous coat which is first involved, so that the surface of the tube appears studded with little white knobs. This I have observed several times. Winckel figures a characteristic

specimen in his handbook. In the largest true cancerous tumour which I ever examined, the tube was deeply sunk between two lobes of the tumour, but quite uninvolved in the disease. Schröder noted that the tube in carcinoma of the ovary remains for a long time intact.

I can find but two undoubted cases of primary malignant disease of the Fallopian tube recorded in recent medical literature, and in one the growth was described as sarcoma. Dr. Martin, of Berlin, informed me in January that he had operated on two cases of carcinoma of the tube, and had given the specimens to his assistant, whose description of them will, I understand, be shortly published. Bruckmüller found medullary cancer in the walls of a bitch's tube. In Scanzoni's case of cancer of the left tube and right ovary, the disease probably began in the ovary, as that organ was "the size of a fist," whilst the tube was "stretched to the thickness of a man's thumb," almost reversing the proportions in the case exhibited this evening. As Scanzoni observed, the case proved that cancer of the Fallopian tubes does not always arise from the contiguity of those organs with diseased neighbouring structures.

In the most recent special treatises on tubal affections by Hennig,[1] Bandl,[2] and Schröder,[3] malignant disease of the tumour is spoken of as though it were without exception secondary, Scanzoni's case being held as doubtful. Dr. Winter[4] has recently described a case of primary carcinoma of the ovary where the tube was obstructed and dilated. A lobulated cancerous mass, almost the size of a walnut, had grown from the ovary, through the tubal wall. It had become polypoid, bearing a thin pedicle and spreading out in the ampulla, the walls of which it had caused to atrophy. The ovarian tumour was the "size of a small fist." As Winckel[5] has already pointed out, secondary cancer of the tube is rare in uterine cancer, as the part of the uterus which is the most frequently attacked is that which lies farthest from the tube.

[1] 'Die Krankheiten der Eileiter, &c.,' 1876.

[2] "Die Krankheiten der Tuben," Billroth and Luecke's 'Deutsche Chirurgie,' 1886.

[3] 'Ziemssen's Cyclopædia of the Practice of Medicine,' vol. x.

[4] "Bericht ueber die Verhandlungen der Gesellschaft für Geburtshülfe und Gynäkologie zu Berlin," 'Zeitschrift für Geburtsh. u. Gynäk.,' Band xiv, Heft 2, 1888, p. 559.

[5] "Lehrbuch der Frauenkrankheiten,' 1886.

The first of the two undoubted cases of primary malignant disease is described by Dr. Senger, of Breslau, in the 'Centralblatt für Gynäkologie,' Jahrgang x, 1886, page 601. The patient, aged 51, died of diabetes, and there were no clinical symptoms of pelvic disease during life. Both tubes were involved, the left more than the right. There were two oval dilatations of each tube, with a less dilated segment between. Papillary masses grew from the tubal mucous membrane, especially in the two dilatations. The largest masses were the least markedly papillomatous. In the left tube there were larger soft growths, not papillomatous, and there was a secondary deposit in Douglas's pouch. Dr. Senger believed that the growths were small round-celled sarcomata. But, in a polypoid growth in one of the dilatations he found "collections of tubes lined with a single layer of cylindrical epithelium and surrounded partly by true sarcoma tissue, partly by new connective tissue rich in nuclei." He stated that the origin of this glandular growth was uncertain, but thought that the gland-tubes might have been derived from the parovarium, which was closely incorporated with the outer wall of this second cyst. It is difficult to understand how parovarian tubes could have forced their way through the walls of the Fallopian tube. The "collections of tubes" rather suggest the appearance seen in my specimen far from the parovarium (Plate XIV, fig. 3). I regret that the account of Dr. Senger's case was published without illustrations. Some readers may not feel certain that the growths were sarcomatous and not carcinomatous. The irregular dilatation of the tubes in this case was remarkable; it should be borne in mind in future researches on the distinctions between suspected sarcoma and carcinoma of the tube.

The second case of primary malignant disease was certainly cancerous. It occurred in Dr. Martin's practice and is described by Dr. Orthmann in the same volume of the 'Centralblatt für Gynäkologie' as Dr. Senger's case, at page 816, and, like that case, is unfortunately published without any illustrations. The patient was forty-six years old and had only been married for three years. About a year and a half before operation she had suffered from typhoid fever, and during convalescence she noticed lumps forming in the abdomen. A firm tumour, "the size of a child's head," was found to the right of the uterus; a smaller swelling was detected to the left, somewhat posteriorly. Ascitic fluid in small amount

was found in the peritoneal cavity. The smaller swelling was caused by encysted serous perimetritis; its clear fluid contents were emptied and drained through the vagina. The right tumour was removed. It consisted of the thickened, tortuous right tube and two pus-cavities. The ostium of the tube communicated with one of these cavities, which Dr. Orthmann described as ovarian abscesses. The mucous membrane of the tube was covered with soft papillary growths, filling the lumen at the abdominal end, where they were numerous. Each growth consisted of a stroma of connective tissue, including numerous nests of epithelial cells. Here and there involutions of epithelium were detected passing into the stroma. The tube, when tapped at the beginning of the operation, yielded "a litre and a half of green pus," no doubt derived from the abscess.

I must here note the evidence, derived from the present case as well as from Drs. Senger's and Orthmann's, that malignant disease of the tube may result from a degeneration of papillomata of the tubal mucous membrane. Dr. Amand Routh informs me that papillomatous material came away in the discharges, when he scraped out the uterus over a year before the operation. In the two cases which I have quoted the growths were partly or entirely papillary. However, I failed to trace any signs of malignancy in either of the specimens of papilloma of the tube which I have exhibited before the Society (October, 1879, and February, 1888), and the first as well as the second case is still living. The first case ('Transactions,' vol. xxxi) resembles Dr. Orthmann's in one respect, for in both the ostium of the tube remained unobstructed so that the escape of morbid secretion caused in the former ascites and hydrothorax, in the latter abscess in the neighbourhood of the ovary. In the case of primary cancer exhibited this evening there was no dropsy, but this complication would have developed had the ostium remained open after the appearance of the morbid growth in the tube. The cancer most probably extended to the ovary, not through the ostium, but between the layers of the broad ligament, which was infiltrated with new growth (fig. 1, *g*). When the attack of inflammation set in, at the end of January, 1887, the tube was possibly studded like the uterus with soft, fungous growths, partially papillomatous, which subsequently underwent malignant degeneration. In this sense, cancer of the tube might represent an extreme and secondary result of chronic disease of

the appendages, of which a more evident result is tubo-ovarian cyst or even papilloma of the tube, as I have already endeavoured to demonstrate in the course of this session (*v.* p. 200). The tubular bodies (fig. 3) might be developed from the deep involutions which abound in papillomatous outgrowths. I have already shown that they could not be of parovarian origin, as Senger suggested in his case.

Whether the growths arose from papillomata or direct from the mucous membrane, I believe that it is most probable that the tubular bodies in fig. 3 were originally developed from the glands of the tubal mucous membrane. As I have already noted, the character of the cancer tends to prove this theory. I have no doubt whatever that Fallopian tube glands exist. Hennig and Bland Sutton[1] have both demonstrated their existence in our species after long and careful observation.

The relation of the appearance of this growth to the menopause is a question of some interest. The last menstrual period was in August, 1887. The patient was forty-eight, but the tumour had already appeared. Hence it is possible that the menopause was hastened through the debility caused by the growth of the tumour. It must be noted, however, that Dr. Senger's case was fifty-one years old, and Dr. Orthmann's forty-six. Considering, then, the ages of all three cases, we may suspect, though we can come to no decision on evidence so scanty, that there is some distinct relation between the menopause and the appearance of malignant disease of the Fallopian tube. *May* 1*st*, 1888.

[1] "The Glands of the Fallopian Tubes and their Function," by J. Bland Sutton, F.R.C.S., 'Trans. Obstet. Soc.,' vol. xxx. "The so-called rugæ of these tubes are really glandular diverticula, whose function is to secrete an albuminous material comparable to the albumen of an egg."

33. *Cyst in corpus luteum.* (*Card specimen.*)

By Samuel G. Shattock.

[With Plate XXXVIII, fig. 1.]

An ovary showing a cyst formed in a corpus luteum. The cavity of the cyst is 1·3 cm. in diameter. Its wall varies slightly in thickness; at its least superficial and thickest part it is ·3 cm., and in the recent state was of the characteristic yellow colour.

The cyst lies close beneath the surface, its situation being indicated by a slight elevation in the general contour. There is a shreddy layer of lymph on the outer surface of the ovary; its interior appears normal.

Since cyst-formation in the corpora lutea of animals was first drawn attention to by Mr. Sutton in the Society's 'Transactions,' vol. xxxvi, in the 'Journal of Anatomy and Physiology,' vol. xx, and in his 'Introduction to General Pathology,' 1886, one or two examples have been observed in the human subject, but none has been as yet placed on record, or figured in the Society's 'Transactions.'

The largest observed by Mr. Sutton (in a cow, out of seventy cows examined) was not larger than a small orange. Nor is it probable that such cysts attain any important dimensions, when their mode of origin is considered, for they can be classed neither as retention-cysts arising in a continuously secreting structure or structure adapted for secretion (as is obsolete gland tissue or an obsolete canal), nor as serous cysts arising in lymphatic tissue.

May 1*st*, 1888.

34. *Case of perfect uterus masculinus with perfect Fallopian tubes and testes in the broad ligament. Complex or vertical hermaphroditism.*

By C. Stonham.

[With Plate XV.]

The very interesting specimen which I bring before your notice to-night was removed from the body of a child aged 9 months, admitted into University College Hospital to undergo an operation for the radical cure of a right oblique inguinal hernia. I am indebted to the kindness of my friend Mr. Horsley, under whose care the patient was, for the opportunity of dissecting and making use of this specimen.

On admission, the hernia could be easily kept up by the finger. The child was in a weakly state, and the testes were undescended.

On August 5th, 1885, the child was put under chloroform, and the operation conducted in the usual way. On opening the sac it was found to contain what seemed to be a uterus with a broad ligament stretching off from each side, and also two organs properly placed for ovaries, and what seemed to be Fallopian tubes. The bladder was not in the sac, but was intimately adherent to the internal ring. This being the case the wound in the sac was sewn up, and the whole returned into the abdomen, the pillars of the ring being afterwards drawn together with catgut. The wound suppurated, the temperature rose, and the child died on August 16th.

At the *post-mortem* examination the following condition was found. On the right side the hernia had again come down, the sac was adherent to the surrounding parts, and the contents to the sac. The accompanying drawing shows the condition of things at the time of the *post-mortem*. The internal organs of generation appeared to be those of a normal female, but subsequent examination showed that what appeared to be ovaries are in reality testes. The uterus did not occupy the middle line, but had been dragged over to the right, owing to the right broad ligament having descended into the hernia sac. Thus the fundus uteri and left broad ligament occupied the right iliac fossa.

On removing the sacrum and greater part of the left innominate

bone, and dissecting out the pelvic organs from the left side, it was found that the uterus terminated below in a vagina ending in a well-formed prostate gland, which surrounded the neck of the bladder. No vesiculæ seminales could be seen, nor at this stage were any vasa deferentia found. I laid open the vagina from behind, and after great trouble succeeded in passing a bristle from the vagina, through the prostate, and along the urethra out at the meatus. There was a well-formed bulb. On making a section of the supposed ovaries I was at once struck with their resemblance to testes, and on submitting them to microscopic examination found them to be so in reality. I then removed the genital organs and bladder, and dissected off all the peritoneum, tracing out the ducts as far as possible. The following conditions then presented themselves:

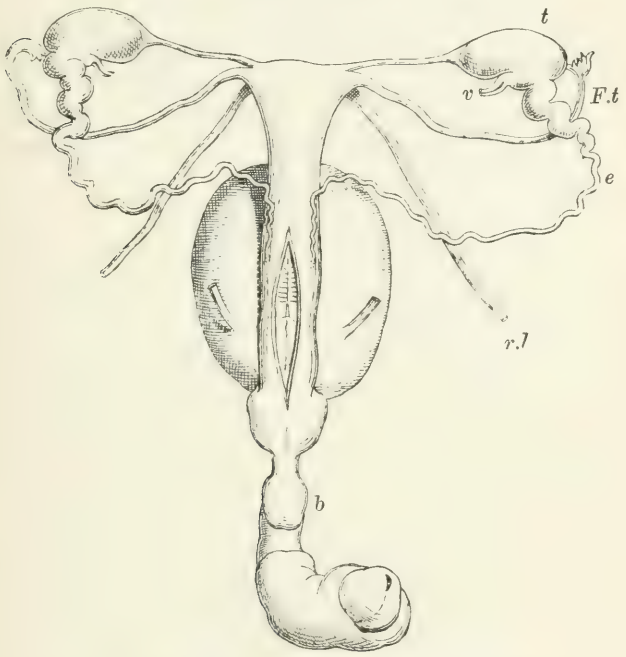
External organs of generation: These were of the male type with undescended testes. The scrotum was deeply puckered, and had a well-marked central raphé. The penis is normal in appearance, but the urethra opens rather far back, constituting a slight degree of hypospadias. The prepuce is deficient at the lower part and envelops the glans like a cowl, so that there is no definite frænum. The corpus spongiosum terminates posteriorly in a well-marked bulb. The membranous urethra is about ·5 cm. in length.

Pelvic organs: The bladder is normal in size and shape, and its neck is surrounded by the prostate gland, which measures 1·5 cm. in its greatest transverse diameter. Projecting from the posterior part of the prostate, and taking a direction upwards and to the right, is the vagina and uterus, with broad ligaments, round ligaments, Fallopian tubes, and on each side an oval smooth body in the position of the ovary but being really a testis. The deviation of the structures to the right is due to the right broad ligament having been dragged down in the hernia. At first sight the case appeared to be one of transverse hermaphroditism, the internal organs being female. The vagina and uterus have been laid open from behind.

The vagina measures 1·5 cm. in length, the interior is traversed by well-marked rugæ, and passing down the anterior wall is a distinct ridge, indicating the original separation of the vagina into two halves by the median antero-posterior septum. At the upper end of the vagina is a small orifice leading into the canal of the cervix uteri, but there is no projection of the external os.

The body and neck of the uterus together measure 4 cm. in length; the lower part of the body is elongated, and there is no sharp line of demarcation between it and the cervical canal, owing to the absence of the internal os. The cervix presents the characteristic arbor vitæ. The interior of the body is traversed by strongly-marked rugæ, and one fold passed along the anterior wall to the cervix, blending here with the arbor vitæ.

Woodcut 10.



v. Vessels. *t*. Testis. *F. t*. Fallopian tube. *e*. Epididymis. *r. l*. Round ligament. *b*. Bulb.

On dissecting out the various structures in the broad ligament I found that the round ligaments were normal. The testis occupies the upper and back part of the broad ligament, and is attached to the corresponding cornu of the uterus by a band of tissue 2 cm. in length, and representing the ligament of the ovary.

Immediately below the testis is the Fallopian tube, which joins the uterus between the round ligament and ligament of the testis.

The free end is fimbriated, and a bristle can readily be passed along it.

On removing the peritoneum from the Fallopian tube a convoluted tube was found in close connection with it. This proved to be the epididymis. It is a good deal convoluted, and measures about 10 cm. in length, extending from the outer end of the testis, where it forms a globus minor, to about the middle of the uterine wall at its postero-lateral aspect, where it becomes so fine that I have not attempted to follow it farther, but I am of opinion that it may run down as a very fine tube as far as the prostate, opening into the posterior part of this. This point could be elucidated by microscopic examination. I may also suggest that there may have been rudimentary vesiculæ seminales in this situation, but at the same time I am bound to say that I dissected the specimen with great care, and could not find any trace of them.

The spermatic vessels are blended together into a cord resembling the round ligament, and are entering the testis after giving off small branches to the epididymis and surrounding parts.

The family history in this interesting case is noteworthy. In some instances I have only the mothers' statements of the facts, and therefore do not attach much importance to them; in other cases I have had the opportunity of examining the subjects themselves. The mother, E. S—, aged 42, has been married twenty-two years, and had fourteen children and eight miscarriages. Seven of these children are dead from various causes, and in none of them was there any question, so far as external configuration goes, of any sexual malformation. Of the living children the two eldest are said to be all right, one a male and the second a female, nine months pregnant when I heard of her. She has, however, a bifid nipple. The third is a girl aged 16. She has menstruated regularly for the last year, and is normally formed. Her mother says that during the menstrual period she bleeds from the nose. The fourth child is a boy, a cripple, and I am indebted to Dr. Pearson for examining him for me, he being under that gentleman's care at the Cripples' Home at Kensington. The external genitals are normal.

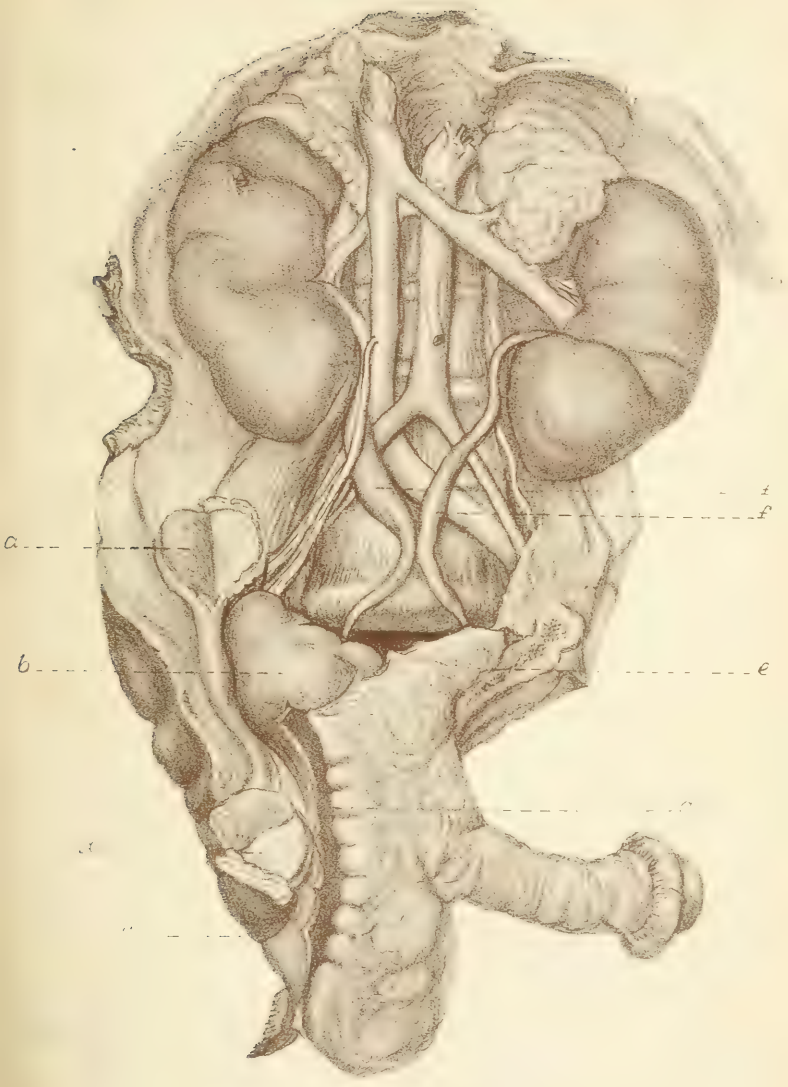
The fifth and sixth children are boys aged 12 and 6 years; they are both well formed and strong for their age. The sexual condition of each is the same. There is a well-formed penis and scrotum but no testes, nor could I detect any fulness in the

DESCRIPTION OF PLATE XV.

To illustrate Mr. Stonham's paper on Complex or Vertical Hermaphroditism. (Page 219.)

From a drawing by Mr. H. Collins.

a. Testis.
b. Uterus.
c. Hernial sacs.
d. Margin of incision with the marks of the sutures.
e. Ramus of pubes in horizontal section.
f. Ureter.



F. Collins del.

E. Burgess lith.

inguinal canals, nor did pressure elicit tenderness. So far the condition is exactly like that of the child from whom the specimen was taken. I did not make a rectal examination as it was objected to.

Dr. Biggs, of Crouch End, is kindly keeping his eye on these boys, so that in the future we may hear more of them, especially should menstruation occur through the penis. The youngest child is a normally-formed female four months old.

Of the father's relations nothing is known. One of the mother's sisters' children has the following history, but I may again say that I have this only on the mother's statement, corroborated by the sisters. With regard to the patient herself I may say that at the Infirmary at which she attended for some minor ailment they remember nothing particular about her. I hope to have an opportunity soon of seeing her myself. This woman, now married, and having had one child, "which was taken from her side," had always been looked upon by her relations as an "hermaphrodite." She is said to resemble a man in appearance. On one side of her external genitals there is "a sort of lump" (to use their own expression), but on the other side she is like a woman. May not this refer to a prolapsed ovary? This woman is said to have passed part of her urine through her umbilicus.

Another niece was born with united labiæ, but these were separated, and she is now all right.

On looking through the literature on the subject of hermaphroditism, and consulting the various journals, I have been able to find some very interesting cases of this variety of so-called complex or vertical hermaphroditism occurring in man; although it is certainly, as far as recorded cases go, more common in the lower animals, especially in goats and dogs.

I shall only refer here to those cases met with in man.

Sir James Simpson, in his classical monograph on this subject, divides all cases of complex hermaphroditism into three classes:

(i) Those in which the general type is female, with a uterus and vesicula seminalis.

(ii) Those of general male type with a uterus, and sometimes Fallopian tubes.

(iii) All cases in which ovaries and testes are found on the same body. My specimen belongs to the second class, and it is to others of this class that I desire to draw your attention.

Speaking of this form Sir James Simpson says: "The uterus

occupies its normal position between the bladder and the rectum. It is sometimes defectively developed and of a membranous structure, and occasionally it is not provided with Fallopian tubes, or, in the quadruped, with cornua. The cavity of the uterus communicates with a vagina that either opens into its usual situation externally, or, as happens more frequently, joins the male urethra. In some cases the vagina is wanting, and the uterus opens directly into the canal of the urethra."

The same author also gives many instances of this monstrosity in animals and man thus:

Columbus found a condition like the one I show to night, but in his case there was no prostate; the vasa deferentia opened into the urethra, and there was hypospadias. Harvey found in an embryo a double uterus with two testes, and a rudimentary penis and prostate.

M. Petit records a case in a man aged 22, which is precisely similar to mine, with the exception that this man possessed vesiculæ seminales to which the vasa deferentia were attached; but, as I previously said, I think this may prove to be the case with my specimen if it is submitted to microscopic examination. The drawing which I have shown you is taken from Förster's Atlas, and shows an example of this condition.

Ackermann mentions the case of an infant in whom two testes were found, one descended but the other in the groin. There was an imperfectly developed uterus and vagina, the latter joining the urethra. The vasa deferentia penetrated the uterine wall at its upper angle in the situation of the Fallopian tubes. They did not, however, open here but passed down in the substance of the wall, and then opened by small orifices into the urethra beyond the point at which the vagina joined it. In my case the vasa deferentia also pass into the wall and become lost, but whether or not they open directly or run down lower microscopic examination can determine.

Steghlener met with a somewhat similar case in an infant. The uterus was distended with fluid; it terminated in a vagina joining the urethra after being dilated into what appeared to be prostate. At the middle of the fundus uteri the vasa deferentia (in this case impervious) were attached. The testes were in the iliac fossa. The canal of the urethra was partly obliterated, and hence the distension of the bladder; the left kidney was cystic.

Mayer has collected five cases, all of which he dissected himself,

and in two of these there was only one testis. In the case of a fœtus with extroversion of the bladder, Mayer found a two-horned uterus and vagina opening into the bladder. The left vas deferens ran to the vagina, but the right one formed the right cornu of the uterus. The vesiculæ seminales were rudimentary, and the glans penis imperforate. In another fœtus Mayer found the usual male organs, but in addition a two-horned uterus and vagina terminating in the urethra.

In another case the testes were found in the inguinal canal. There was a uterus, vagina, and Fallopian tubes, which passed with the testes into the canal. The vasa deferentia and vesiculæ seminales opened into the vagina. In the two cases in which Mayer only found one testis both were adults. In one there was hypospadias with a perineal fissure communicating with the urethra in front and the vagina behind. The vagina was nearly three inches in length, and had a uterus at the upper end. A well-formed prostate was present, and close to the right inguinal ring a testis; there was no testis on the left side. On each side there was a vesicula seminalis opening into the vagina. On both sides a canal was found, but whether this was Fallopian tube or vas deferens could not be determined owing to previous mutilation of the parts.

In Mayer's other case the testis was on the left side. It had an epididymis and vas deferens. There was a prostate and two vesiculæ seminales. The ejaculatory ducts opened into a vagina which was present with a uterus, the former passing through the prostate and joining the urethra. The penis was fissured along the corpus spongiosum, the meatus opening at its root.

Professor Langer has recorded a case ('Brit. and For. Med.-Chir. Rev.,' vol. i, 1856, p. 270) of a man, aged 63, who had been married thirty years but was childless. On dissection, a bi-corned uterus masculinus was found ending in two Fallopian tubes, one at each cornu. The testis and epididymis on each side was embraced by the ala vespertilionis. On the left side the uterus was drawn over by a scrotal hernia. The uterus ended in the prostate. Vesiculæ seminales were absent.

With regard to the occurrence of more than one hermaphrodite in the same family, assuming that the two boys to whom I have referred may present a similar condition to the one I show, I have found an interesting case recorded by Sir Everard Home in his work on 'Comparative Anatomy,' vol. iii, p. 321. A Devonshire

woman had had three children, of whom the second one was a normally formed girl; but the other two were considered to be hermaphrodites. Both of them were idiotic, stunted in growth, and unusually fat, and both presented the same sexual peculiarity. The mons veneris was loaded with fat, there was no penis but a prepuce in one case one sixth of a inch long, and in the other one inch long; under this prepuce was the meatus. There was no vagina. There was an imperfect smooth scrotum, and an indented line in the situation of the raphe. The scrotum contained two small testes. One of these children had also a supernumerary finger on each hand and a supernumerary toe on each foot.

In conclusion, I am anxious to direct your attention to the occurrence of hernia in this case. I do not think that this is an accidental condition, but am inclined to view it as the natural outcome of the tendency the testes manifest to descend into the tunica vaginalis.

Had this child lived I think it quite probable that at the time of puberty menstruation might have occurred through the penis, although I do not feel by any means sure on this point since there are no ovaries. The secondary sexual characters would have been unquestionably male.

I desire to thank my friend Mr. Donald Gunn for the excellent drawings which he has kindly made for me, and also Dr. Wallis, of East Grinstead, for making the microscopical sections of the testis and epididymis. *February* 21*st*, 1888.

VI. DISEASES AND INJURIES OF THE OSSEOUS SYSTEM.

1. *Deformity of sternal ends of both clavicles.*

By Walter G. Spencer, M.S.

Annie L—, aged 2 years, Chadwick Ward, Westminster Hospital, under the care of Mr. Macnamara. She has well-marked evidences of past rickets, besides the deformity of the clavicles.

The mother states that the wrists and ankles became deformed when the child was about eight months old. About the same time the clavicles began to be deformed, the deformity gradually becoming more marked. Within the last six months the child has had whooping-cough. It was quite healthy at birth. Its diet has always been good. None of the other children has had rickets. There is no trace of syphilis.

The drawing (exhibited) gives the appearance of the clavicles as seen from the front. At the inner end of the prominence a firm mass is to be felt between the clavicle and the first rib, attaching it thereto and to the sternum.

The child was seen by members attending the Society's meeting. Opinions differed as to the conditions really present. Broadly, these are divisible into two :—(1) That the deformity has been the result of an enlargement of the sternal end of the clavicle, or of a firm mass pushing the clavicle forwards; (2) that it was a sharp rectangular bend, close to the inner end of the clavicle, due probably to the contraction of the sterno-mastoid muscle, and of the nature of a green-stick fracture. To the latter view it may be objected that the deformity is exactly symmetrical, and no callus can be felt, whereas greenstick fractures are generally unilateral, and a good deal of callus is thrown out.

The clavicle generally bends about its centre, and this seems much more likely than a bend within half an inch of the end of

the bone. However, Dr. Penrose has shown some greenstick fractures at the angles of the ribs, in which scarcely any external callus has been formed. If contraction of the sterno-mastoids can have caused a sharp bending of the bones it cannot have been done during the recent whooping-cough. The child may have had some bronchitis when the rickets came on which the mother has forgotten. But then bending of the clavicles does not ordinarily follow even severe bronchitis in rickets.

The exact symmetry and the sharp prominence would appear to point to the sternal end of the clavicle having been pushed forwards by some rickety changes either in the head of the clavicle or in the cartilage of the first rib or in the sterno-clavicular interarticular cartilage, and comparable to the process which forms "beads" on the ribs. I need hardly say that the epiphysis does not appear until the twentieth year, and is then only insignificant.

I have to thank Mr. P. M. Yearsley, of the Westminster Hospital, for the drawings. *February 7th*, 1888.

2. *Isolated fracture of base of skull.*

By J. H. Targett, M.S.

[With Plate XVI, fig. 1.]

This specimen was taken from a lad, aged 18, who on March 11th, 1888, was boxing with another boy, when the latter slipped and fell, striking the patient on the left mastoid region with his head. After the blow he was very giddy, and had to be supported into the house. He vomited several times, and it is said that a little blood and watery discharge flowed from the left ear. The following day he had a slight headache, which grew worse, and towards night he lost consciousness, ultimately becoming violently delirious. The delirium subsided on March 13th, but he was still unconscious when brought to the hospital on that day. He has suffered from chronic otorrhœa on the left side for some years.

On admission.—A thin ill-nourished lad; temperature 101°, pulse 68, regular, moderately strong. Slight tenderness over left mastoid region. No otorrhœa, perforation of left membrana

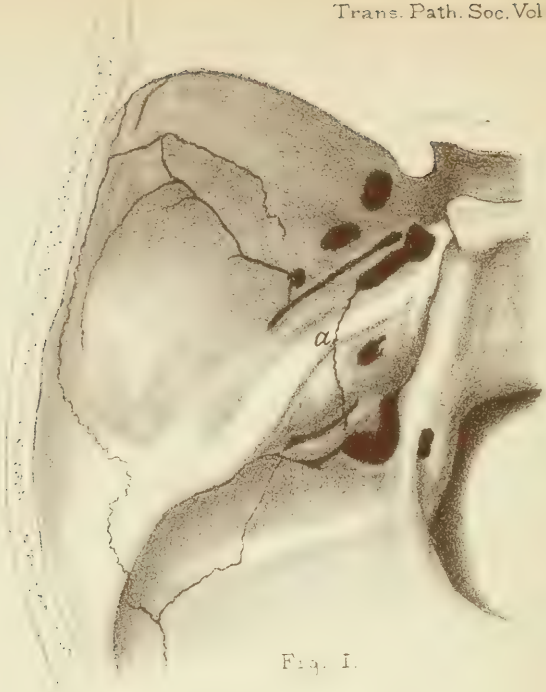


Fig. 1.

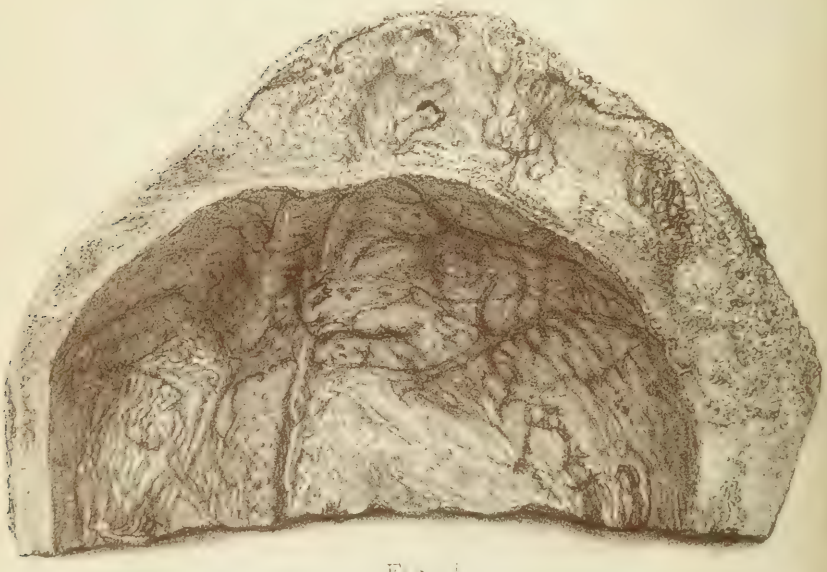


Fig. 2.

E. Burgess lith.

DESCRIPTION OF PLATE XVI.

Fig. 1 illustrates Mr. Targett's specimen of Isolated Fracture of Base of Skull. (Page 228.)

Fig. 2 illustrates Dr. Jacob's specimen of Myxoma of the Skull. (Page 249.)

From a photograph.

tympani. Pupils equal and active; conjugate deviation to left; discs healthy. Lies in an unconscious state. There is some weakness of face and arm on right side. Urine passed involuntarily. Heart and lungs normal.

March 15th.—Distinct right facial paralysis; and loss of power is marked, though not complete, in right arm. Temp. 102·8°.

17th.—To-day the patient shows signs of returning consciousness, and his eyes follow the movements of those around him. Temp. 103°, pulse 114, respirations 58. Pupils equal.

18th.—The semi-conscious state continued, the pulse became more rapid and fluttering, and he died early this morning, just a week after the accident. There were never any convulsions.

Autopsy.—There was general suppurative meningitis, and the brain was considerably softened, especially around the lateral ventricles; but there was no obvious reason for the hemiplegic symptoms. After removal of the dura mater a fissure-fracture was discovered running across the left petrous bone obliquely forwards and inwards (Plate XVI, fig. 1). It was entirely isolated, extending from the groove for the lateral sinus to a point just behind the foramen spinosum, and crossing the petrous bone slightly external to the internal auditory meatus. The posterior wall of tympanum was remarkably thin and fenestrated, doubtless resulting from the old ear disease, while its cavity was partly filled with clot which extended from the neighbouring sinus. This clot must have resulted from a slight laceration of the lateral sinus produced by the fracture, for there was no general thrombosis of the sinuses, nor suppuration in the vicinity of the fracture. The whole of the lower lobe of left lung was completely collapsed and airless. Other viscera normal.

Remarks.—The chief points of interest in this specimen are the *direction* and *limitation* of the fracture, as they tend to support von Wahl's theory of the production of fractures of the base of the skull ('Centralblatt f. Chirurg.,' December, 1887). Mr. Arthur Hare has recorded ('Lancet,' February, 1888) a somewhat similar case, and he points out that these isolated fractures of the base are of great importance in that they may be regarded as the first steps in the building up of the more ordinary types of fracture "where the complexity of the lesion is apt to mask the manner of its production." He is also of opinion that they are much more frequent than is generally supposed. *May 15th*, 1888.

3. *Compound comminuted fracture of the lower jaw, with rupture of the inferior dental nerve.*

By Walter G. Spencer, M.S.

An Irishwoman, aged 50, was admitted into the Westminster Hospital, August 1st, 1887. She had met with a fracture between the first and second molar teeth on the left side, from direct violence. There was a good deal of swelling of the cheek; the fragments were in good position. She complained very much of pain, which became limited after a time to the distribution of the inferior dental nerve. As the pain diminished there was found to be anæsthesia over the dental part of the inferior dental nerve. After six weeks she became an out-patient, still complaining much of pain. The second molar tooth was loose in the line of fracture, but she would not have it extracted for some time. Before this tooth was extracted the large piece of bone exhibited came away, viz. on October 10th. On October 17th the second piece came away.

On October 31st she had anæsthesia over an area bounded by a line drawn vertically from the angle of the mouth, and extending to the middle line. This included the outer surface of the lip as far as the lower border of the jaw, the inner surface of the lip, and the outer surface of the gum, the incisor, canine and bicuspid teeth. Union appears to be progressing, but another piece of bone threatens to come away. Pain has almost ceased.

The larger piece of bone exhibited is part of a vertical section, a quarter of an inch thick, showing the outer and inner surface, and the canal for the inferior dental nerve. The smaller piece, also a portion of a vertical section, is pierced likewise by the canal.

The inferior dental nerve was apparently torn at the time of injury, and the presence of a tooth assisted in causing separation of the comminuted portions.

Remarks.—The inferior dental nerve is only exceptionally injured. A large number of the fractures are in front of the mental foramen. When the fracture is between the inferior dental foramen and the mental foramen, the nerve is said to generally escape because the displacement is not sufficient. In this case there was no dis-

placement when the patient was seen first, whatever there might have been at the time of injury. There was no bleeding from the inferior dental artery. The comminuted fragments have been broken off vertically in the same line as the principal fracture. To the patient the chief matter was the excessive amount of pain which the irritation of the proximal end of the nerve caused. There were no changes in the skin of the cheek beyond the bruising. *November 15th*, 1887.

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4. *Displacement of the head of the femur, through a fracture of the acetabulum, into the pelvis.*

By Timothy Holmes.

The following case, and the preparation accompanying it, will, I think, be found of considerable surgical interest in connection with the case reported long since by Mr. Moore, of the Middlesex Hospital, in vol. xxxiv of the 'Medico-Chirurgical Transactions.' For the notes, and for the accurate description and delineation of the preparation, I am indebted to Mr. C. Stewart Pollock, one of our students.

T. S— was admitted to the surgery of St. George's Hospital at nine o'clock on the evening of November 29th, 1887, having fallen from a loft sixteen feet on to his left side. He was suffering from fracture of the lower end of the left humerus, and complained of great pain in the left side of the pelvis. The patient, who never rallied, died of collapse at 5.40 a.m. on the morning of November 30th.

Diagnosis on admission.—The greatly collapsed condition of the patient prevented any rigorous examination being made.

1. Elbow: Crepitus was obtained on flexion and extension of the forearm, but none on supination and pronation; movement was free. On the inner side of elbow was a loose fragment giving crepitus; on the outer side, at a higher level, was a prominence which was taken to be the external condyle; it was immovable. There was no movement obtained on grasping the prominence and

the shaft of the humerus. The distance from the prominence to the olecranon was shorter by a quarter of an inch than the distance from the internal condyle to the olecranon. The flexor surface of the elbow-joint was normal. The arm was put up on an internal angular splint in the best position.

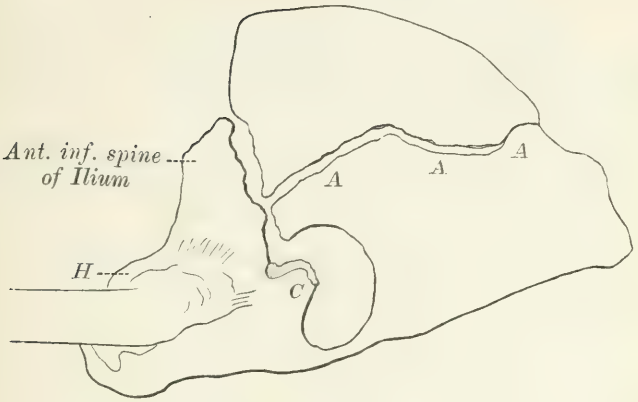
2. Left lower extremity: The leg and the foot were not everted; the thigh was easily flexed on the pelvis, marked crepitus being obtained while doing so. The measurement from the anterior superior spine of the ilium to the internal malleolus was shorter by one inch than the same measurement on the right limb. The great trochanter was deeply sunk and impinged upon, but was not markedly above, Nélaton's line. Voluntary movement of the limb was absent, but there was free passive movement. No further attempt at diagnosis was made owing to the great collapse.

Diagnosis.—Intracapsular fracture of neck of femur.

Post-mortem examination.—L. humerus: There was a great deal of extravasation round the elbow-joint, and at the back, where there was a contused wound. The lower end of the bone was fractured in two places, the inner condyle and ulnar articular surface constituting one fragment, the capitellum and outer condyle constituting the second fragment. The *inner fragment:* The line of fracture extended from one third of an inch above the condyle, and passed at first obliquely upwards and outwards to the centre of the anterior surface of the shaft, one inch above the coronoid depression, then passed vertically downwards to the interval between the ulnar and radial trochlear surfaces, where a clean vertical fracture separated this fragment from the capitellum, the fractured surfaces looking, the *upper*, forwards and downwards, the *lower*, upwards and backwards. The *outer fragment:* The line of fracture extended from one third of an inch above the external condyle nearly horizontally inwards to join the descending line of fracture of the internal condyle. The anterior ligament of the joint was torn, the upper fragment being driven between the two lower fragments so that the internal border of the shaft of the humerus looked forwards.

The left os innominatum: There was very extensive extra-peritoneal extravasation, extending on the left side as high as the kidney (the organ was not ruptured), and on the right side as high as the true brim of the pelvis. The left acetabulum had been fractured, the head of the femur presenting in the pelvis to the

Woodcut 11.



Front view.

Woodcut 12.

Back view.

extent in size of half-a-crown. The ligamentum teres was uninjured, and the capsular ligament whole.

The ilium: This bone is fractured in three places.

1. (Woodcuts 11 and 12, A.) The line of fracture extends from half an inch in front of the posterior superior spine, at first downwards and forwards for one inch, then forwards for an inch and a half, then directly inwards to about a quarter of an inch from the linea ilio-pectinia.

2. (Not shown in the Woodcut.) The anterior inferior spine was torn away obliquely at its upper part, but directly at its lower and inner part.

3. (Woodcut 12, B.) That part of the ilium which helps in the formation of the acetabulum was fractured from the rest of the bone, and forced upwards.

The ischium is fractured in four places.

1. (Woodcuts 11 and 12, C.) The back part of the body is torn away, including the spine. The lines of fracture extend (α) from the anterior and upper part of the great sacro-sciatic notch, at first forwards, separating it from the lower and forward part of the venter of the ilium, (β) downwards, and then obliquely backwards, separating it from the anterior part of the body.

2. (Woodcut 12, D.) The front part of the body is separated behind from the posterior part of the body by the line of fracture (β). Above it is separated from the body of the pubes by an irregular line of fracture (γ); at its upper part is the fragment.

3. (Woodcut 12, E.) A small quadrilateral piece of bone, forming the groove for the obturator vessels and nerve.

4. (Woodcut 12, G.) This fragment consists of the ascending ramus of the ischium and part of the descending ramus of the pubes, the line of fracture extending across the outer part of the ramus of the ischium, in front of the tuberosity.

The pubes is broken into three fragments.

1. (Woodcuts 11 and 12, H.) Consists of the horizontal ramus, the back part of the ilio-pectineal eminence, a small portion of the ilium, that part behind the anterior inferior spine (δ), the crest, angle, spine, and commencing portion of the descending ramus. The outer end of this piece (δ) presents a sharp spinous projection, where it has been torn away from the ilium.

2. (Not shown in the Woodcut.) Consists of the ilio-pectineal eminence, and that part of the pubes which forms one fifth of the

acetabulum. The edges of this fracture are very ragged and serrated where it is torn away from the horizontal ramus, but it appears to be a clean fracture where it is separated from the anterior inferior spine of the ilium.

3. (Woodcut 12, G.) This fragment, noted before, consists of the descending ramus of the pubes and the ascending ramus of the ischium.

The sacro-iliac synchondrosis is not injured.

As illustrating the same surgical injury I also produce a preparation from St. George's Hospital Museum, which I found, when Curator of the Museum, in the body of an old man who had died after what was supposed to be a fracture of the cervix femoris. The femur is uninjured, but the acetabulum is traversed by several lines of fracture running through the pelvic bones, and the head of the femur would have been forced into the pelvis but for the persistence of a bridge of bone crossing the gap in the acetabulum.

The chief point in practical surgery connected with this injury is the possibility of diagnosing it from a fracture of the neck of the femur. I did not see the subject of the present paper during life, and his collapsed condition would have rendered any detailed examination unjustifiable, but I conceive that the sunken condition of the trochanter might put the surgeon on the right track, together with the extensive crepitation which must accompany so great a fracture of the pelvis. This position of the trochanter is also found in the inward dislocation of the hip, but then the symptoms are quite unlike those of the injury we are considering.

Another interesting point to surgeons would be the possibility of reducing the head of the bone to its natural place, but I hardly think this Society is the place to discuss that subject.

December 20th, 1887.

5. *A rare form of fracture of the lower end of the femur.*

By William C. Bull, M.B.

In fractures of the lower end of the femur a considerable number involve the condyles and extend into the joint. Of these by far the most common are those usually called intercondyloid. A diagnosis is generally easy. In one case recorded, the femoral condyles were fractured transversely in front and behind, so as to form three portions,—a middle one continuous with the diaphysis, and two large splinters, one in front, and one behind, the central part.

The present specimen has just been added to the museum of St. George's Hospital, and presents some peculiarities.

The specimen was removed by amputation from a cabman, aged 56. He was admitted with a fracture of the femur and hæmarthrosis. Suppuration of the joint ensued, necessitating amputation of the limb. Crepitus was felt near the joint, but the nature of the fracture could not be made out.

It will be seen, both in the specimen and the accompanying drawing:

(1) That the anterior portion of each condyle, together with the anterior and internal part of the lower third of the shaft, has been broken off and lifted away from the remainder of the bone.

(2) That the continuity of the bone-fibres passing from the posterior part of the shaft of the femur into the condyles is intact, so that there is no complete solution of the continuity of the bone.

(3) That there is a fine fissure between the condyles, not sufficient to allow one condyle to be moved on the other.

The fracture has united with slight displacement of the anterior fragment upwards; the union is fibrous and fairly firm. There is some destruction of the articular cartilage. The tibia and patella were not injured.

No history could be obtained of the nature of the accident, the man being found underneath his cab. From the specimen, however, it is probable that the force which caused the fracture was applied to the joint surface, *i. e.* that the man fell with his knee flexed and struck the internal condyle against the ground. A

similar condition to this is seen more often in the patella, when it is fractured on its joint surface, by the condyle of the femur being driven against it. In such a case the fracture does not always extend through the whole thickness of the bone.

December 20th, 1887.

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6. *Simple comminuted fracture of the head of the tibia.*

By D'Arcy Power, M.B.

The specimen which I show to-night would have formed, I had hoped, a fitting pendant to the interesting preparation which Mr. Bull has just exhibited. It does not do so, I find upon a more careful dissection than I was able to give it when I placed a notice of it in the hands of Dr. Coupland, for I then believed it to be an example of a simple longitudinal fracture of the head of the tibia.

The fracture is, however, a sufficiently rare one to warrant my trespassing for a short time upon your patience. It is an impacted comminuted fracture of the upper end of the tibia, with a longitudinal fracture extending upwards through the internal tuberosity into the knee-joint.

The patient was a woman, aged 76, who fell down twenty stairs, and was picked up with her leg doubled under her. She survived the accident for a fortnight. I found, on dissecting the tibia, that she had sustained a comminuted fracture of the tibia with impaction. The fibula is also broken to pieces just below its neck. The lower fragment or shaft of the tibia has been driven upwards and forwards into the head of the bone, whilst a longitudinal fracture runs across the articular surface of the internal tuberosity. The line of this fracture is single until it reaches the posterior border of the tibia, where it divides into two branches, one of which runs directly backwards, whilst the other runs outwards. A small portion of the articular surface of the external tuberosity is split off by this bifurcation.

The main interest of the specimen lies, I think, in the fact that it shows very clearly the way in which a longitudinal fracture

occurs in these cases. In the example before you there can be no doubt that it has resulted from the impaction of the lower into the upper fragment. The force producing the fracture appears to have been applied through the articular surface of the head of the tibia, by the patient falling head first downstairs and striking her bent knee in the descent.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series iii, No. 990 (*a*). *December 20th*, 1887.

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7. *Dupuytren's fracture of the fibula.*

By J. Hutchinson, jun.

Although the term Dupuytren's fracture has been for long well recognised, examples of it are so uncommon that I thought the following case worthy of record, the specimen being a typical example of this variety of fracture.

Michael R—, aged 25, was admitted into the London Hospital under the care of Mr. Waren Tay with a Dupuytren's fracture of the left leg, due to the following accident. He was engaged in unloading a vessel of stones, each mass of stone weighing upwards of two tons. He was packing one upon the other when one fell over, and as he jumped aside to avoid it his left foot was caught and crushed by the falling weight. On admission, there was a large wound extending from one inch above the ankle over the dorsum of the foot to the base of the toes, a large flap of skin being reflected, so that the peroneus brevis was fully exposed, and the joint between the os calcis and cuboid could be felt to be opened. The internal malleolus was fractured as well as the fibula in the lower fourth. The foot was dislocated outwards and upwards so that the astragalus lay between the shafts of the tibia and fibula. This malposition was readily corrected before he was taken to the ward.

Primary amputation was advised, but refused by the patient. Both anterior and posterior tibial arteries appeared to be uninjured.

The limb was kept in a bath of a solution of corrosive sublimate, but the skin sloughed extensively, and the temperature rose to 105°

owing to free suppuration. On the thirteenth day after the accident amputation was performed about the middle of the leg by long anterior flap; suppuration was found to have extended into the tissues at the seat of amputation. Owing to this the flaps healed slowly, and there was a sinus remaining at the time of his discharge, four months after admission. By the great increase of width of the ankle, the projection of the tibia and the ascent of the foot, it was possible in this case to diagnose Dupuytren's fracture, a diagnosis which was fully confirmed by examination after the operation. The specimen exhibited shows that the internal malleolus was completely detached from the tibia and went with the foot; the external malleolus was also broken across at its broadest part, but the small fragment was not displaced. Higher up, about three inches from the end, the bone was also broken somewhat obliquely, and the lower fragment was bent right away from the tibia, having dragged away with it a narrow border of bone, the attachment of the interosseous and inferior tibio-fibular ligaments. The interosseous membrane was torn at its lower fourth, but higher up was intact, and therefore prevented any gap occurring between the shafts of the tibia and fibula. Mr. Henry Morris, writing in 'Holmes's System' (vol. i, p. 1092), says that "Dupuytren thought the upward displacement of the foot was due to separation of the fibula from the tibia, but his own and other preparations seem to prove that the upper part of the fibula keeps its position to the tibia." Whilst fully agreeing with this view I am not aware that Dupuytren has published any description of any preparation of the fracture which is known by his name. I have carefully searched in his works and find accounts of two cases, in neither of which was the specimen obtained, nor could I find any illustration of it in the Musée Dupuytren at Paris. However, the three specimens, Sir Astley Cooper's, in St. Thomas's Hospital Museum, Mr. Thomson's, figured in the 'British Medical Journal' for 1880, vol. i, p. 919, and my own which is brought from the London Hospital Museum, are identical in their main points. In each the fibula has been broken within three inches from the ankle, and whilst the lower fragment has been displaced outwards, the rest of the shaft has retained its normal position. This can also be seen in the life-size drawing accompanying M. Dupuytren's original paper ('Annuaire des Hôpitaux,' vol. i, 1819). It is interesting to note that in two out of the three the tibia has yielded rather than the extremely strong

interosseous ligament, affording an example of what is met with in fractures of the patella and some other bones, that when a violent strain falls upon bone and ligament together the former breaks, as a rule, rather than the latter yields. Dupuytren thought that the tibio-peroneal ligaments gave way. What was the exact condition in Mr. Thomson's case is difficult to say, since the specimen was obtained three years after the accident, when anchylosis between the tibia and astragalus, as well as between the fibula and astragalus, had occurred. The history is briefly as follows:—A woman, aged 65, slipped in the road and fell. The left tibia was dislocated inwards, and projected through the skin, the fibula was broken about an inch above the malleolus, and the foot dislocated outwards and upwards. The dislocation was reduced, but severe sloughing followed, and suppuration extended up nearly as far as the knee-joint. After a long illness the patient recovered with the original deformity persisting. The foot was everted and the ankle immovable. Finally, as the foot was almost useless, Mr. Thomson performed Hey's amputation.

As regards the rarity of the occurrence of Dupuytren's fracture, we have that surgeon's authority for the statement that he found it only once out of 200 cases.

During three years of my term as surgical registrar at the London Hospital there were 350 cases of fracture of the fibula, including 140 cases of Pott's fracture. In this time, besides the case I now place on record, there was one other which was perhaps an instance of Dupuytren's fracture, so that the proportion would agree with that given by M. Dupuytren. It is, however, more interesting to note that as compared with Pott's fracture it occurs in the proportion of about 1 to 100. This fact bears witness to the immense strength of the connection between the tibia and fibula at their lower ends.

M. Dupuytren's original case, the first which was described with care, is reported in the 'Annuaire Médico-Chirurgicale des Hôpitaux' of 1819. The patient came under his care forty-three years after the accident, and so extraordinary was the deformity that M. Dupuytren caused a life-size representation of the limb to be made. It is in the Atlas attached to the 'Annuaire,' and has been copied by Mr. Thomson in his paper in the 'British Medical Journal.' From this drawing one can readily see that the fibula has been fractured two to three inches above the end of the

malleolus, and that the lower fragment is united at nearly a right angle with the long upper one, the outline of which is indicated as parallel with the tibia. The foot has been displaced outwards and upwards, and also rotated somewhat outwards, so that the leg was shorter than its fellow, and at the level of the ankle much broader, about twice as wide as the normal, so far as one may judge from the drawing. A line drawn from the projecting lower end of the tibia (the malleolus of which had been evidently broken off) to the summit of the angle formed by the two fibular fragments, indicated exactly "the centre of movement which had led to the displacement of the foot," in other words the case differed from an ordinary Pott's fracture in that the foot had gone up to a certain extent on the outer side of the ankle as well as being rotated outwards.

No movement was possible at the ankle, but the patient was able to make fair use of the limb.

The accident was as follows:—He was engaged in excavating, when a mass of earth gave way, and he fell a distance of twelve feet with the weight upon him. Great swelling followed, and the hæmatomata having been opened, suppurated freely, some necrosis subsequently occurring. These complications interfered with proper treatment of the fracture-dislocation, and no doubt explained the great subsequent displacement to which M. Dupuytren draws attention.

The same surgeon published another case in his 'Leçons Orales,' vol. i, 1839, p. 369, which was by subsequent writers confused with the one already quoted, and the drawing copied by Mr. Adams in the 'Cyclopædia of Anatomy and Physiology,' and by Mr. Thomson in the 'British Medical Journal,' has been erroneously supposed to belong to the following case. A man, aged 54, when coming out of a public-house, half drunk, fell down an incline with his right leg everted and sustaining the whole weight of the body. He was carried to the Hôtel Dieu, and there, besides the usual signs of Pott's fracture, it was noticed that the leg was shortened, and the distance between the two malleoli almost doubled, that the astragalus, lower fragment of the fibula, and with them the rest of the foot, had ascended upwards of two inches above the normal position. At first efforts at reduction failed, but after copious blood-letting they were successful, and the limb was adjusted on an inside splint. On the next day, whilst the apparatus was being

re-applied, sudden muscular spasm reproduced the deformity, but reduction was again affected after flexing the knee and distracting the patient's attention from the foot. He subsequently made a perfect recovery.

Dupuytren's fracture may be said to consist in a Pott's fracture with separation of the tibia and fibula, and the method of its production is almost identical. By forcible eversion of the foot the outer ridge of the articular surface of astragalus is driven against the angle formed by the external malleolus with the tibia, whilst at the same time the outward pressure against the malleolus tends to tear it away from that bone. Hence, in addition to the wedge-like action of the astragalus, we have the traction exerted through the fibula. It seems probable that the fibula is forced away from the tibia before the two bones break. Mr. T. Bryant and Sir W. Fergusson each record a case of separation of the two bones with impaction of the astragalus between them, impaction so firm that no effort was successful in reducing it. It must, however, be noted that the diagnosis of each case rests on clinical evidence alone.

Of the four cases now mentioned of Dupuytren's fracture, two were due to falls in the street with the foot strongly everted; in the other two a weight fell upon the foot.

In the London Hospital Museum is another specimen of fracture of the external malleolus with extensive rent of the inferior tibio-fibular ligaments, but as there was evidently no marked gap between the two bones, and hence no ascent of the foot, it would perhaps not be right to count it as an example of Dupuytren's fracture. *May* 15*th*, 1888.

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8. *Separation of the upper epiphysis of the radius.*

By C. Mansell Moullin.

This specimen came from a boy 16 years of age, whose arm was crushed by a machine accident. The external condyle of the humerus is broken into two or three pieces, and there is a deep depression in the capitellum leading into a fissure; the upper

epiphysis of the radius is completely detached, the line of separation passing between the cartilage of the extremity and the diaphysis; otherwise it is not injured, though the orbicular ligament is torn away. The synovial membrane of the joint still retains its connection to the head of the radius. Amputation was performed a short distance above the elbow-joint, owing to the extensive laceration of the soft parts in the forearm. The hand was quite uninjured, the whole of the violence falling upon the outer side of the elbow and forearm.

The chief interest attaching to this specimen is its extreme rarity. Stellate fractures of the head of the bone detaching portions of its circumference are not uncommon; but, so far as I am aware, no injury of this kind has been presented to the Society, and no case is mentioned in Billroth and Lücke's 'Surgery.' The diagnosis is an exceedingly common one. There is a peculiar accident of very frequent occurrence in infants and young children from the way in which they are lifted about by the hand, or swung round by the arms; it is not attended with any displacement, but there is a certain amount of indistinct crepitus to be felt, and there is a considerable degree of pain and of inability to use the limb. This is very often said to be due to separation of the epiphysis, but no proof of this has ever been brought forward, and there can be little doubt that in the majority of instances at any rate it is really the result of the head of the bone slipping from underneath the orbicular ligament. *November* 15*th*, 1887.

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9. T-*shaped impacted fracture through the lower epiphysis of the femur, with bony union.*

By C. Mansell Moullin.

The patient from whom this specimen was obtained was admitted into the London Hospital at Christmas, 1886. About a year before, when he was fifteen years old, he sustained a compound fracture of the lower end of the femur. From this he recovered completely, and was able to get about again fairly well, with a slight amount of deformity and a certain degree of stiffness about

his knee-joint. A few days before admission this stiffness began to increase, and he found not only that he was unable to bend the joint but that the attempt was exceedingly painful. On examination, the popliteal space was found to be occupied by a tense swelling, in which a slight degree of pulsation could be detected, especially on the inner side. There was a scar on the front of the limb; the joint could not be bent to a right angle, and even that was exceedingly painful. A faint bruit could be heard at times where the pulsation was most distinct; the limb itself was fairly well nourished, but colder than the other, though the tibial arteries could scarcely be felt. Pulsation was checked at once by pressure on the femoral, but no change of any kind could be produced by direct pressure.

The diagnosis, which lay for a long time between myeloid sarcoma and arterial hæmatoma, was settled in favour of the latter by complete cessation of the pulsation on digital compression. An incision was made in the popliteal space rather to the inner side of the middle line; a very large quantity of recent coagulum turned out; and the end of the upper fragment found projecting backwards and pressing upon the popliteal artery, in which there was a small opening with rounded edges, evidently formed by gradual ulceration. The vessel was isolated as well as it could be, and tied above and below, the projecting fragment being removed by means of a chain saw. For three weeks everything went on as well as could be, but about the fourth the knee-joint began to enlarge, the wound gaped open again, though it was all but healed, and a great mass of granulating tissue made its appearance. Finally the joint suppurated, and the limb was amputated.

After maceration the epiphysis was detached and the line of fracture could be followed on the posterior surface of the shaft, along the intercondyloid notch, and upon the anterior surface of the epiphysis. Evidently the condyles had been separated and were united again; but while the cartilage still persisted between the epiphysis and the shaft over the whole of the rest of the surface, along the line of fracture it was replaced by bone, and this had to be broken through before the epiphysis could be detached.

November 15*th*, 1887.

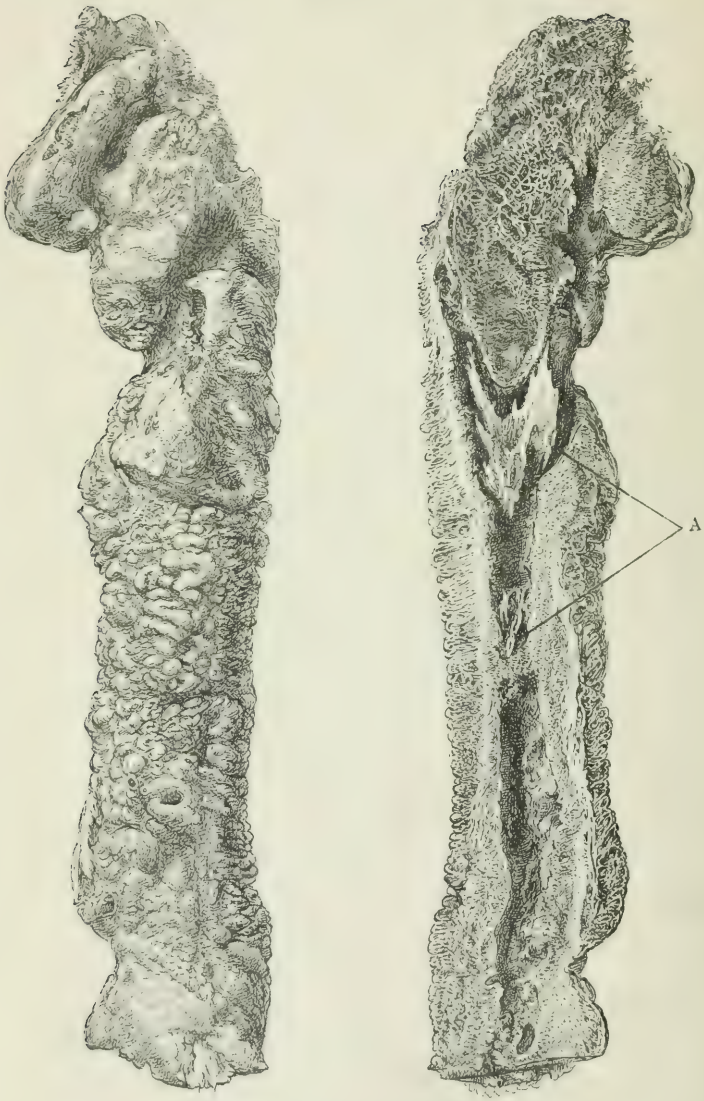
10. *Cases illustrating chronic osteitis, and chronic osteo-peri-osteitis, due to central necrosis.*

By Robert William Parker.

Case 1.—The specimens depicted in Woodcut 13 were removed from a boy, aged nearly 10 years. An injury of no great severity was followed by a large extra-periosteal abscess involving the greater part of the thigh, associated with severe general constitutional symptoms. After an incision had been made, the femur could be felt running through the centre of the abscess cavity, but no bare bone could be discovered. In the course of some weeks, however, a small superficial sequestrum from the popliteal surface, and a long thin spicule apparently from the medullary cavity, were removed with slight temporary benefit. As the hip- and knee-joints had become subsequently affected it was determined to amputate at the hip-joint. Upwards of ten months had elapsed since the first commencement of the disease. For some weeks the severe constitutional disturbance had seemed to contra-indicate so grave an operation, but as the boy lived on, and as the local condition appeared to get worse rather than improve, the operation was finally undertaken. After removal of the limb the bone was found covered with periosteum except at a narrow interval at the lower part of the posterior surface of the shaft, just above the condyles, and over an oval patch a little below the smaller trochanter. The shaft was thickened, as compared with the opposite side; the bone was heavy. The head of the femur was detached. The periosteum was very firmly adherent, and could only be separated after considerable maceration; its exterior surface was soft and covered with florid exuberant granulation tissue; but it was still tough and resistant.

After removal of this periosteum, the surface of the femur was found much roughened from a deposit of new bone; this new bone was soft, very vascular, and firmly incorporated in the texture of the femur. The femur was sawn through vertically as seen in the woodcut, p. 246. At the upper spot, uncovered with periosteum, it now presented a central necrosed piece of bone (A), apparently the outer shell of the compact tissue of this part of the femur. In the remains of the medullary cavity (which was full of inflamed

Woodcut 13.



A. Loose sequestrum.

and semi-purulent marrow) were found several smaller pieces of necrosed bone, probably the remains of the interior compact layer. The whole femur is enlarged. Very little compact substance remains; for the most part this has become changed, and merges into the roughened subperiosteal deposit elsewhere referred to. The compact tissue can, however, be distinguished, and the changes more especially due to the periosteum can be traced, and differentiated from those which have occurred as obviously in the bone itself. The interstices between the new deposit of bone were filled with soft granulation tissue; their honeycomb appearance, depicted in the plate, did not show itself in the recent state of the bone, and has only become manifest since maceration and drying. The bone now feels comparatively light.

Case 2.—As in the former case, the disease followed a slight fall, which set up some slight synovitis of the knee, of a chronic kind. The patient was a boy aged 9 years. Four months after the fall painful induration of the soft parts of the thigh, with localised thickening of the shaft of the femur just above the condyles, came on. Pus was let out, but no bare bone could be discovered. Eight months later, *i. e.* twelve months after the initial injury, the femur was greatly thickened in its lower two thirds; a sinus led down to the bone, but bare bone could not be felt. This sinus was enlarged, the bone exposed, and a small trephine introduced; a fragment of necrosed bone was found and removed. But no real improvement took place, and after some further delay, the boy being ill and exhausted, the limb was amputated in the upper third of the thigh.

On examination of the bone, after its removal from the surrounding soft parts, the periosteum was seen to be normal; it peeled off the bone with the greatest readiness, except above and around the trephine hole. On sawing through the femur, in a vertical direction, a (second) fragment of necrosed bone was discovered, in the centre of the shaft. In its recent condition, the bone was heavy and solid; the cancellous spaces were filled with granulation tissue, blood-vessels, extravasated blood, and pus. As now exhibited in the dry condition (after maceration) the bone is almost uniform in appearance; there is no medullary cavity, and no appreciable compact layer of bone; the whole femur appears to consist of cancellous bone; the lower epiphysis is in a similar condition, and has grown *pari passu* with the shaft; the line of junction with the

shaft does not present any abnormal appearance. The shaft is at least twice its natural size, the increase being greater in the lower part than in the upper; the amputation was made through a part slightly thickened.

The contrast with the preceding specimen is very marked. In the former there is less general enlargement, and the changes are most marked on the surface of the bone; in this specimen, the surface of the bone is almost normal, while the shaft is greatly thickened and consists almost entirely of cancellous bone.

In connection with the preceding cases may be mentioned another, that of a boy, aged 10 years, who without known cause began to suffer pain in the left leg (and also in the shoulder). The pain was slight at first, but became very severe, and was thought to be acute rheumatism, and for three weeks previous to his admission he was treated for acute rheumatism.

On admission into the East London Hospital for Children, his thigh was seen to be greatly swollen; there was no redness of the surface, but the superficial veins were turgid; the foot was œdematous, the limbs very painful. An anæsthetic having been administered, incisions were made and about 40 oz. of pus were evacuated. The femur was found bared of its periosteum over a considerable area. The abscess continued to discharge large quantities of pus for some weeks. As the boy's health was rapidly deteriorating, the limb was removed.

After separating the soft structures from the bone, the periosteum was found to be detached from the greater part of the surface of the femur. There was no new bone either on the surface of the femur or on the periosteum the inner surface of which was covered with soft velvety granulation tissue. At the points where the periosteum again came into contact with the femur there was considerable proliferation of new bone, to which the periosteum was firmly adherent. The bared bone was dry and whitish in colour, and on vertical section was seen to be unaltered both in texture and in size. The medullary cavity contained semi-purulent medulla. In brief, the exposed part of the bone was dead, and showed no reparative power. The periosteum, though detached from the bone, was not dead, owing probably to its connection with the adjoining soft parts; it is to be noted that it showed no bone-producing power, except where it remained in contact with living bone.

Remarks.—The specimens appear to me of some interest as

raising the question how far the pathological processes displayed are the result of an osteitis or a peri-osteitis. In the first case, there is a considerable amount of new bone on the surface of the femur, which may not unfairly be attributed to the periosteum. This deposit of bone, however, is the more remarkable when it is borne in mind that the periosteum was cut off from its connection with the soft parts around, and that any pathological process that may have occurred must have got its blood supply from the bone direct. Although I could not find any bare bone when the abscess was first opened, I think it very probable that the bone was bare near the smaller trochanter at the spot where the necrosed portion is seen. It is interesting to note the fact that at this spot no new growth has taken place, either osteal or peri-osteal. The periosteum was everywhere greatly thickened; its exterior surface was covered with flabby exuberant granulations, its interior surface was so firmly adherent to the bone that it could not be separated until after considerable maceration.

In the second case the bone itself appears to have been chiefly affected; the whole bone is converted into cancellous tissue, and the medullary cavity obliterated. In the recent state this cancellous tissue was filled with young granulation tissue and vessels and was very vascular. The periosteum seemed quite unaltered; it everywhere peeled off the bone with the greatest ease, except just around the opening made by the trephine, where there was a little roughness and irregularity of the surface.

I would just draw attention to the state of the bone, so-called osteo-porosis. This condition is an artificial one, due to maceration, and the consequent removal of the blood and granulation tissue which fill the interstices during life. The occurrence of this condition may always be taken as a sign that chronic inflammation has existed at some time during life; the amount of porosity fairly indicates the intensity or chronicity of the process.

In both these cases the source of irritation was the central necrosed piece of bone. The fragment of bone was killed or injured by the accident alluded to in the clinical notes, and not being cast off set up the chronic osteitis and inflammatory hypertrophy by means of which the fragment gradually became more and more deeply embedded. In the first case there was in addition an extra-periosteal abscess which irritated the periosteum and led to the outpouring of the new osteoid tissue seen on the surface of the femur.

In Case 2 there was only a small amount of subperiosteal new growth, apparently the result of the surgical interference, for at the time of the operation the periosteum readily peeled off the bone, the bared surface being quite smooth and normal. On the other hand, the bone was greatly thickened, the changes being chiefly in the interior of the bone. In Case 3 the periosteum was widely separated from the bone by pus, and although it retained its connection with the surrounding soft parts, there were no indications of periosteal new growth. The femur likewise showed no signs of repair, owing doubtless to the completeness and extent of the necrosis. It would thus appear as if repair in such cases depends on vitality in the bone itself. Case 1 points out that such part as the periosteum plays in this repair depends on its being in contact with living bone. Case 2 indicates clearly that very considerable new growth may go on in a bone, and its periosteum meanwhile remain almost quiescent; while Case 3 appears to teach that the periosteum alone cannot initiate the process of repair.

On the other hand, there are many clinical facts to show that bone can repair without the aid of periosteum; for instance, in the case of wounds of the skull or of the superficial surface of the tibia (as situations where the process can be conveniently watched) considerable areas of bone may be denuded of periosteum and be repaired notwithstanding; the denuded bone assumes a dry whitish aspect; after an interval of time, the whiteness gives place to a vascularity, which, beginning as puncta, spreads and soon covers the dead area; granulations then appear, and finally such portions of the necrosed bone as are not re-vitalised or absorbed are raised from the living bone beneath and thrown off.

But if a diseased bone be shelled out of its sheath of periosteum, either by the outpouring of pus or by surgical means, before any new bone has been formed, as a rule, the bone will not be reproduced. Some three months ago, I amputated at the hip-joint for hip disease associated with a very chronic osteo-myelitis of the femur. I proceeded to amputate the limb through the middle of the thigh by means of a circular incision. Having tied the vessels, I next made a longitudinal incision on the outer side of the limb, dividing the periosteum down to the bone, and then shelled out the remains of the femur as high as the hip-joint. This was accomplished with great ease, and, with a view to keep open the sheath of periosteum and to stimulate its osteogenic function, I inserted a

large india-rubber drainage-tube. Although the child has made an excellent recovery, and the wounds have all closed, I cannot say that any real bone has been found in or by this periosteal sheath, that is to say, I could not detect any new bone with the probe when I finally withdrew the drainage-tube. There was a long stump, and its centre was quite firm, but I think not bony. It will be remembered that a similar plan was adopted by the late Mr. Shuter, and that a Committee of the Clinical Society (*vide* 'Transactions' vol. xvi), appointed to consider whether new bone had been formed from the periosteum left in the stump, replied in the negative.

I contend therefore that, contrary to text-book teaching, in such cases as these, repair depends almost exclusively on processes which must commence in, and be largely carried on by, the bone itself; and that the importance and powers of the periosteum have been exaggerated.

Considering that bone forms the foundation on which the body is built up, that it is highly specialised in structure and composition, is exceedingly vascular and richly supplied with nerves, it is not a little remarkable that so large a share of function, both in health and disease, should have been attributed to the periosteum, which by comparison is but an adventitious structure and chiefly useful in affording a ready means for the attachment of muscles.

February 7th, 1888.

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11. *Acute necrosis.*

By A. Quarry Silcock, M.D.

[With Plate XVII.]

The specimens shown are those from a case of infective osteomyelitis, apparently starting in isolated inflammatory foci situate at the line of junction of the epiphysial cartilages with the diaphyses of the tibiæ, and terminating in acute necrosis.

Matilda G—, aged 11, was admitted into St. Mary's Hospital on August 31st, 1887, by reason of pain and some swelling of the left leg. The pain was localised to the head of the tibia of that side,

and intense tenderness was experienced on pressure over the inner tuberosity and adjacent portion of the diaphysis. There was some tumefaction of the soft parts in this region, and a slightly marked redness of the skin with increased heat, but no sense of fluctuation on palpation. The patient was a thin, pallid, sallow-faced girl, although her cheeks were somewhat flushed; her temperature was 104·6° F., the pulse 108. She was nervous, restless, and cried out greatly when her leg was touched.

The family history was good; she bore no evidence of tubercle or syphilis, and had suffered no previous illness, except an attack of measles a few years since. The mother said the child had been playing on the pavement the day before the pain below the knees began, and she had said to her "she would get cold if she did not take care." When the pain did begin, that is on the 28th, or two days before admission, it began suddenly, the child remarking, "Oh! mother, I have got a pain in my knee." It never ceased afterwards.

The day after admission, the leg was more swollen and painful. I saw her, and thinking that I had to deal with a case of epiphysitis, I directed that a longitudinal incision should be made over the upper epiphysial line on the inner side, antiseptically. This was done by the house surgeon Mr. Crowle, but he found no pus.

On September 2nd—the next day—the patient appeared to be in less pain, the highest recorded temperature being 102°, and when the dressings were removed a small quantity of curdy pus oozed up from the depths of the incision.

On the 5th, she complained of pain and tenderness about the head of the *right* tibia, but there was no swelling or redness of the parts.

On the 6th, the surfaces of the wound were found to be coated with a thick diphtheritic-looking membrane; this was pulled off, the incision slightly enlarged, and a small quantity of pus—a drop or two—liberated from beneath the periosteum, just below the epiphysial line, by passing the director in various directions through the original periosteal incision, for that membrane had previously been freely incised.

On the 7th, the tenderness below the *right* knee was more marked, and a fulness, which was thought to exist below the inner tuberosity on the day before, still more apparent; as in the case of the other leg, there was, however, but little redness, and no discov-

erable sense of fluctuation. The temperature on the evening of the 6th had risen to 103·2°. I made a longitudinal incision down to the bone, and after separating the periosteum in the region of the epiphysial line, ultimately set free a small quantity of curdy pus. The wound was dressed with iodoform and alembroth wool, after being freely irrigated with ·1 per cent. solution of perchloride of mercury.

On the 9th, the soft parts about the *right* inner malleolus became œdematous, and marked tenderness on pressure over that prominence apparent.

On the 10th, an incision was made down to the right inner malleolus, and the covering periosteum found to be inflamed, but there was no collection of serum or pus beneath. It is noteworthy that this wound subsequently entirely healed up, although, as may be seen from the specimen, there was actually a small focal abscess just above the epiphysial line in the centre of the lower part of the diaphysis. Similarly, there had appeared to be considerable pain on pressure over the *left* inner malleolus, but as it seemed to decrease in intensity subsequently, the parts were not disturbed.

The further history of the case may be very briefly related. In spite of every effort to procure adequate drainage by means of counter-openings, one being made in the popliteal space of each side, the periosteum became extensively separated from the upper fourth of the diaphysis on each side, and both knee-joints became full of fluid.

On the 30th, that in the right knee-joint was found, after aspiration with a hypodermic syringe, to contain a large number of leucocytes, to be sero-pus in fact; consequently I laid the joint open by free lateral incision. The cavity of the joint was irrigated night and day, but as the suppuration spread, and the condition of the leg became daily more hopeless, Mr. Page, in whose absence I had charge of the wards, amputated the right thigh on October 5th, at the junction of the lower and middle thirds. The effusion into the left knee-joint subsided; the necrosis of the left tibial diaphysis, however, steadily extending. It was thought useless to attempt the removal of the necrosed shaft, since there was no chance of a useful limb resulting, the regenerative power of the osteogenetic layer of the periosteum having been destroyed by suppuration so acute, intense, and widespread; hence amputation was advised. This procedure the parents would not agree to, and as an alternative

Mr. Page, on October 26th, removed the whole of the left diaphysis, piece by piece, through a long longitudinal incision. A few bony spicules and plates attached to the inner surface of the detached periosteum alone attested feeble regenerative efforts to form new bone.

The patient gradually improved in health; but it will be observed that the amount of new bone thrown out to replace the diaphysis of the left tibia is quite inadequate.

The right tibia, which was sawn through longitudinally, shows that the inflammatory process is focussed, as might have been expected from the clinical facts which I have detailed, at the upper and lower ends of the diaphysis, in those regions which French authors have called *juxta-epiphysial*, or, as I would term them, if such a nomenclature is desirable, *para-epiphysial*—that is to say, in the region where the processes of growth in the direction of length and ossification are being carried out. The upper epiphysis is partly separated from the shaft, and beneath the upper epiphysial cartilage are several small sequestra, embedded in what microscopically may be seen to be pus. This focus of suppuration has extended upwards into the epiphysis itself, the central portion of the epiphysial cartilage having been absorbed, and through the right half of the epiphysis by a small sinuous channel into the knee-joint, having perforated the right articular cartilage, in which a small hole may be observed. Burrowing outwards along the line of the epiphysial cartilage, it has reached the periosteum, raising this, and so denuding a considerable portion of the upper third of the shaft. To the naked eye the medulla of the middle third or more of the diaphysis appeared to be healthy. At the lower end, the suppuration is not so extended, but here too, in the para-epiphysial zone, may be seen a yellow patch, which, as seen in the microscopical sections, proves to be pus; like that at the upper end, it encloses small sequestra, and has partly absorbed the epiphysial cartilage on its way to invade the lower epiphysis.

The microscopical examination of the bone does not materially modify or add to one's conception of the characters of the lesions as they are apparent to the naked eye. The bipolar foci of inflammation and suppuration are not circumscribed, for the cancellous tissue of the neighbouring portion of the diaphysis and epiphysis are infiltrated with inflammatory cells, that of the epiphysis, however, in a far less marked degree than that of the diaphysis. The histological characters of the lesion are quite unlike those associated

Fig. 1.

Fig. 3.

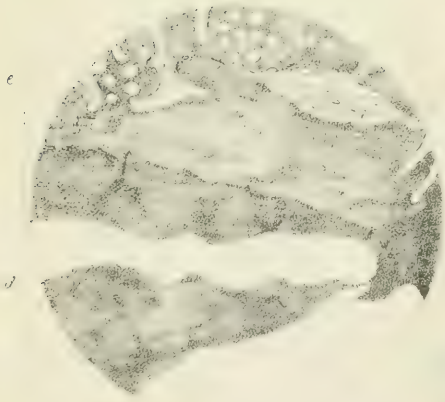
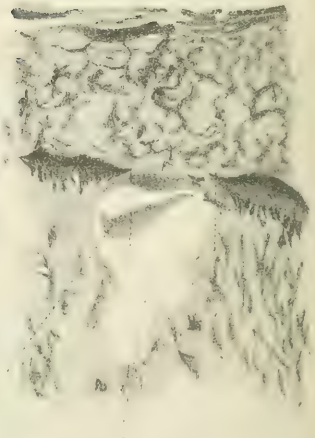


Fig. 2.



DESCRIPTION OF PLATE XVII.

To illustrate Mr. Silcock's case of Acute Necrosis. (Page 251.)

From drawings by H. C. Ridley and R. Ostlere.

FIG. 1.—Longitudinal section through para-epiphysial region of lower end of right tibia.

d. Diaphysis showing invasion of suppurative focus.

e. c. Epiphysial cartilage, which at one point is perforated.

e. Epiphysis showing inflammatory infiltration most marked opposite the focus of suppuration in the diaphysis.

FIG. 2.—Longitudinal section of para-epiphysial region of upper end of right tibia, magnified seven times, showing perforation of epiphysial cartilage, and cavity in diaphysis from which a sequestrum has dropped out, leaving a small quantity of purulent matter.

FIG. 3.—Upper margin of cavity and adjacent portion of epiphysis (× 50) where the epiphysial cartilage has been perforated.

with the tubercle bacillus, so that the latter may be safely excluded; they seem to demonstrate infection from the two primary centres of the diaphysis to the epiphysis, to the periosteum, and to the medullary cavity. It is interesting to note what confusion has been carried into the serried ranks of cartilage cells of the epiphysial line by the advancing inflammatory process; they are proliferating, but in hopeless disarray, and disappear as they are engulphed in the dense host of small round cells. These are crammed into the osseous areolæ, and so tend to strangulate the vascular loops which are essential to the process of ossification, and compass the destruction of that spongy layer which is not yet bone, but is no longer cartilage, uniting the epiphysial cartilage to the diaphysis (Plate XVII).

I have called this disease acute necrosis at the suggestion of Mr. Beck, to whom I showed the specimens and related the case, and there can be no doubt that inflammatory trouble beginning in such situations has a great tendency in young subjects to spread widely, to the epiphysis, the periosteum, and the medulla. Ollier remarks, "The inflammations which are produced in the spongy tissue on the border of the connecting cartilage, take the form of osteomyelitis, and differ from osteomyelitis of the central canal insomuch as they cannot progress as rapidly, stopped as they are by the walls of the vacuoles of the spongy tissue. They differ from them above all in that they may easily show themselves under the periosteum, from which they are separated at the edge of the cartilage by only a thin layer of compact tissue which is easily medullised. Thus the frequently spontaneous issue of pus from beneath the periosteum, coming from the cavities of the spongy tissue, is explained." The marrow of the spongy tissue is directly continuous with that of the central canal, and of course is continuous with that of the epiphysis when the connecting cartilage is perforated; so panosteitis results.

The symmetry of the bony lesions implies, as Sir James Paget has insisted, a morbific condition of the blood, and the morbid material therein contained enters into combination with the part diseased. The altered state of the blood consisted in a deterioration of quality accompanying the lowered vitality of the patient, and probably, in the presence of a coccus capable of endowing a local inflammation with infective qualities. The para-epiphysial regions in long bones, and the sutural edges of the flat bones of the

face and skull, where the process of ossification is being actively carried on, are peculiarly liable to become inflamed, probably because the physiological balance is easily upset, by strain or other injury, or exposure to wet and cold. So the organism finds a fitting resting place, perhaps because the current through the dilated vascular loops which I have already alluded to, is necessarily slow, as Neumann has suggested, perhaps because the vitality of the cells of the part is easily exhausted, working away as they are at bone making; be this as it may, here the inflammatory process is particularly ready to show itself, and given the advent of a suitable micro-organism, a permanent focus of infection is set up, which may not only lead to death of the greater portion of the involved bone, but become the starting-point of general infection and pyæmia. *March 6th*, 1888.

12. *A case of acute epiphysitis occurring in both knee-joints, left temporo-maxillary joints, and metacarpo-phalangeal joint of right index finger.* (*Card specimen.*)

By John R. Lunn.

R H—, aged 1 month, was admitted into the Marylebone Infirmary, December 27th, 1887, with her mother, as she was at the breast.

The mother stated that her child was born at the full time, was healthy, and there was no trace of swelling about her. After the child was vaccinated her arm was very red and much swollen.

Nine days ago a swelling appeared on the face, and has been getting bigger ever since. A second and third swelling appeared on the right hand and left knee four days after the first. Mother and father both healthy.

Present condition.—On the left side of the face over the ramus of the jaw, is a globular, tense, fluctuating swelling, the skin over it being shiny. It extends on to the cheek, as far as the angle of the mouth, limited above by the zygomatic process of the temporal and below by the body of the jaw (lower border).

On the right hand, over the heads of the last two metacarpal

ones (third and fourth fingers), is a round, red swelling, presenting the appearance of strumous dactylitis. This swelling also fluctuates.

At the inner side of the left knee over the upper epiphysis of he tibia, is another globular swelling. The skin over it is thin and shiny. The right knee-joint seems to be also somewhat enlarged and to contain fluid.

January 2nd.—Under chloroform Mr. Lunn opened the abscesses. That by the knee-joint led down to bare bone in a very ragged condition. That over the jaw discharged some greenish pus when opened, and the temporo-maxillary joint seemed quite disorganised. Both wounds were dressed with iodoform and the leg put on a splint.

The mother was taking Mist. Pot. Iod. ʒj (gr. v) and Liq. Hyd. Perch. ʒss.

3rd.—Wounds quite sweet and look well. The child takes food and sleeps well.

There has been no rise of temperature. Child suddenly got worse and died.

Autopsy twenty-four hours after death.—Body well nourished. In right pleural cavity was a quantity of purulent fluid due to the bursting of an abscess, surrounding the carious body of the seventh dorsal vertebra.

Suprarenal capsules were cystic, each containing one large (dermoid) cyst. Other organs healthy.

Left knee: Old sinus led down to the upper epiphysis of the tibia, which was surrounded by thick greenish pus. The upper part of the tibia was necrosed. No perforation into joint, which was apparently healthy.

Right knee-joint was distended with pus of an unhealthy kind. The cartilage was perforated, and the perforation came from the epiphysis of the femur, which had necrosed. Tibia healthy. In left knee the upper epiphysis of the tibia was affected. In right knee the lower epiphysis of femur was affected.

May 15th, 1888.

13. *Femur, with newly-formed bone, from a case of pyæmia in an infant.* (*Card specimen.*)

By John H. Morgan.

The cartilage covering the head of the femur was separated from the neck, which had been almost entirely eaten away as well as the great trochanter. The periosteum from the shaft was easily stripped off. The upper fifth of the shaft was bare and rough; the next two fifths was covered anteriorly by a thin layer of newly-formed bone forming a shell about 1 mm. in thickness. The lower two fifths had no such covering, and the lower extremity showed hæmorrhage into and inflammation of the cancellous tissue. The periosteum was red and thick and smooth on its inner surface. The hip-joint was entirely destroyed, the knee joint healthy.

The patient was a male, aged 4 weeks, small, thin, and tender ever since birth. A swelling was observed over the upper part of sternum soon after birth, which increased. The child was brought to the Hospital for Sick Children on account of swelling of the left thigh.

Abscesses were opened over sterno-clavicular articulations and left buttock, but the patient died five days later.

At the *post-mortem* examination, the shaft of the right radius was absolutely bare, lying in an abscess of which the periosteum formed the outer wall, and the epiphysis at each end was separated from the shaft. The elbow and both sterno-clavicular articulations were destroyed. Abscesses were found beneath the scalp and upon the surface of the brain, in the left ankle and the right knee. An abscess was found in the liver, and a large vein containing pus in the suspensory ligament. *February 7th*, 1888.

14. *Case of osteitis deformans showing a want of symmetry and lengthening of some of the bones.*

By H. H. Clutton.

This case is brought before the notice of the Society on account of the following features. The deformity is in the upper extremities unsymmetrical, and shows an increase of length in some of the affected bones.

Jane K— is a married woman of 65 years of age.

Family history.—Her mother died of "cancer of the womb" at the age of 43; of her father she knows nothing. Her parents had a large family, the first six members of which died quite young. One of the brothers died at the age of 21 of consumption, one of cancer at 53, and another of heart disease at about the same age. One brother and one sister alive and well.

Previous history.—At the age of two she met with an accident which caused permanent stiffness of left elbow. With this exception she had no deformity of any kind until over 50 years of age. She married at 25 and had four children; no miscarriage.

History of her present deformity.—When a little over 50 years of age she first noticed a slight prominence of the lower end of the right ulna. After a year or two, the right radius became curved; then the left humerus and finally the lower extremities began to show signs of deformity.

During the last three or four years she has diminished in stature, and for the last two or three years her friends have noticed an increase in the size of her head. At times she used to suffer a great deal of pain in the left arm especially at night, but during the last few years she has been quite free from this trouble.

In 1878 she was in St. Thomas's Hospital under Dr. Ord, on account of a swollen ankle and a general eruption. The latter was "black and blue" as if she had been bruised. Ulcers and scabs formed which kept her in hospital for seven weeks. She has had a similar eruption several times since, each attack being slightly less severe than the last. For one of these she was during 1883 under the care of Dr. Stephen Mackenzie, who has very kindly supplied me with many of the above particulars.

Present condition.—Her height is now 4 ft. 6 in. She thinks that formerly it was 5 ft. 1 in.

The lower extremities are seen to be much curved outwards and slightly forwards, and that in the erect position when the feet are together the knees are several inches apart. Both femora and tibiæ are much thickened.

The spine has a great increase in its dorsal curvature and is quite fixed, so that the body is much bowed forward with the chin overhanging the sternum. A few of the highest cervical vertebræ are, however, quite movable, and the ribs move in a natural manner on deep inspiration.

The clavicles are scarcely altered but may be a little thicker than natural. The skull looks a great deal too large in proportion to the face, and measures twenty-two inches in its largest circumference. It will be seen therefore from the above description that this patient has the ordinary features of osteitis deformans, and were there nothing more to describe it would be scarcely necessary, in the present state of our knowledge, to trouble the Society with the notes of this case. But the upper extremities are the parts in which the deformity reaches its height, and it will be seen that it is here unsymmetrical.

The right humerus is straight and of normal size. The left humerus is very much thickened from one end to the other, and has a large anterior curvature occupying the whole length of the bone. In measuring along the *arc* of this curve the left is still half to three quarters of an inch longer than the right humerus. One would expect if there were no increase in the length of the bone that the left humerus would be the shorter of the two, especially as the tape was drawn along the arc of the curve.

The left elbow was anchylosed from accident or disease in childhood. This might possibly account for the increased length of the humerus, as we know that such lengthening sometimes occurs from disease in early life at or near an epiphysial line. But, as will be seen in the description of the forearms, this explanation cannot account for what appears to be an increase in length of the right radius. The right radius is very much curved and thickened, especially in its lower third, whilst the ulna is of normal size and quite straight. The hand is pronated and the carpus partially displaced from the radius and ulna. The curve of the radius is directed outwards and forwards, whilst the hand is displaced inwards and backwards. Consequently, it appears as if the radius had increased

in length, for otherwise the hand would not be displaced in the opposite direction to the curvature of the radius, the curve alone reducing somewhat the length of a bone. Yet notwithstanding its curvature the radius looks as if it had increased in length and pushed the hand towards the ulnar side. The left forearm appears to be normal in every respect.

On careful inquiry I cannot find any explanation of the curvature of the bones of the upper extremities. She is not bed-ridden and therefore does not lean on her arms in attempts to sit up in bed. During the day she is either moving about the house or sitting in a chair.

The absence of symmetry in this particular case has enabled a comparison to be made between the affected bones of one upper extremity with the unaffected ones of the other. The increased length thus demonstrated in the upper extremity rather suggests that the disease is, as Sir James Paget originally thought, an ostitis even in its earlier stage, and not atrophy. In looking over the cases recorded in the Pathological and Clinical Societies' 'Transactions,' since the papers first published by Sir James Paget in the 'Med.-Chir. Transactions,' the ages of some of the patients belong to that period of life when chronic ostitis is not at all uncommon. For example, Dr. Stephen Mackenzie's patient, a woman, was aged 48, and had had the disease five and a half years. Mr. Treves's patient, a woman, was also aged 48, and had had the disease five years. Mr. Morris's patient, a man, was aged 42, and had had the disease twelve years. Mr. Howse's patient, a man, was aged 37, but in this case the bones were not bent, although he looked upon his case as probably one of osteitis deformans.

Out of the last seven cases recorded by Sir James Paget in the 'Med.-Chir. Trans,' vol. lxv, five are reported to have noticed their first symptoms when over fifty years of age. Of the remaining two, however, one was forty years of age, and the other twenty-eight when the first signs of deformity were noticed.

If the disease in its earlier stage were considered as one of rarefying ostitis in which the bone, although spongy and soft, may be larger than normal, the explanation given by Mr. Lunn of an early atrophy and subsequent hypertrophy would be unnecessary. For the varying conditions that may be found in simple chronic ostitis would be quite sufficient to account for all the phenomena.

The microscopical characters also support this view, for in Dr.

Sharkey's report of Mr. Lunn's specimen it is stated that "rarefying ostitis appears to be the predominant process," and that "there is also clear evidence of a formative ostitis." The same opinion is also clearly expressed by Mr. Butlin in his description of the microscopical characters of Sir James Paget's specimens.

The increase of length in some of the bones at this period of life which the absence of symmetry has enabled me to demonstrate comes as an additional confirmation of this view, that the disease is really of the nature of chronic ostitis. This case may, I think, also throw some light upon those instances in which only one bone shows general enlargement and curvature, such as the case recorded by Mr. Bowlby ('Path. Soc. Trans.,' vol. xxxiv, p. 193). For if this patient had not shown general signs of the disease, but only the unsymmetrical enlargement and elongation of some of the bones of the upper extremities, there would have been the same difficulty in proving the case to be one of osteitis deformans.

March 6th, 1888.

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15. *Osteitis deformans, associated with lymphomatous nodules in the spleen.*

By James F. Goodhart, M.D.

These specimens, a part of the calvaria, of the spine, and the spleen, are from a man of 65 years, who was for some time under the care of Mr. Roots, of Kingston-on-Thames.

Originally a post-boy, he had had to do with horses all his life, and in consequence, I suppose, he was of the extremely slim build and small stature characteristic of that employment, but his head was enormous, and suggested his disease at a glance. He had enjoyed good health till about four months before his death, when he began to lose his strength and appetite, and also to emaciate. I saw him on four separate occasions, and was never able to find any positive disease except that in his bones, and we therefore inferred that there must be some visceral cancer.

He told us that for years (about fifteen he thought) his head had been growing larger, and the hats which he had worn in his earlier years would now only lodge on the top of his head. A year ago its circumference, measured by Mr. Irwin Palmer, was twenty-two and a half inches. He had not noticed that he had grown shorter.

He was a wrinkled old man with an enormous, rather bossy head, measuring in circumference twenty five and a half inches, so that it had gained three inches in about a year.

There was very little of him, and he was apparently a good deal emaciated; his back was curved, his thighs bowed outwards, his tibiæ curved forwards. His clavicles also were much curved, but I could not say that any of his bones were enlarged except those of the skull.

His thorax was laterally compressed, and he moved its walls feebly.

There was some resistance in the epigastrium, but with this exception, and the feeble movement of his lungs, the position and the working of his viscera were normal.

The autopsy was made by Mr. Roots, Mr. Irwin Palmer, and myself. The skull was heavy, soft, and thick, three quarters of an inch in some parts. The clavicles were thickened, and the first rib also. All down the front of the bodies of the vertebræ new soft porous bone had been deposited, and the bodies of many of the vertebræ were rarefied and some thickened. The alæ of the ilia were also thickened, and bulged into the iliac fossæ to a moderate extent. The long bones, although some of them altered in curve, were not obviously diseased, but the inspection was made late in the evening and under difficulties, and in this respect not thoroughly.

There were many small fleshy tubera, apparently of a cancerous nature in the liver, and several firm, fleshy, cancer-like glands in the mediastinum and abdomen. There were one or two small nodules of growth in the pleura.

But the chief peculiarity was that the spleen was full of large, circumscribed yellow masses, and presented the typical appearance of the hardbake spleen of Hodgkin's disease.

The lungs, heart, thyroid, stomach, gall-bladder, urinary bladder, prostate, kidney, and intestines were all healthy, and no obviously primary source of growth could be discovered.

The yellow masses in the spleen consist of some obscurely fibrillated, almost structureless material with Malpighian corpuscles on their outskirts, which from their appearance give the impression that they are taking part in the same process of degeneration.

The masses are more fibrous than the general run of tubercular formations, but I do not think that they are more so than the lymphomatous nodules in the spleen of Hodgkin's disease when these have attained any large size, and I think they are of lymphomatous nature. They are certainly not sarcomatous.

The nodules in the liver were all fleshy and free from any tendency to caseate, but I have to regret that the specimen, and also that of the fleshy glands, were lost or left behind at the time of the inspection.

The case is, however, worth record for the curious but interesting association of Hodgkin's disease of the spleen with osteitis deformans. This association may have been a mere coincidence, but I am the rather inclined to think that it is another evidence of the inveterate tendency to cell growth that exists in this peculiar disease, osteitis deformans.

As regards the nature of the splenic tumours, however, I would say this, that I am not altogether sure that the appearances here described may not be common to several distinct growths.

I had not purposed to say anything specially directed to the intimate nature of osteitis deformans, but the remarks of Mr. Clutton upon his case lead me to say that I doubt the soundness of the conclusion that the disease is a rarefying ostitis because of the frequent asymmetry of the disease or its localisation to one bone. I admit the fact—my own case is an evidence thereto—but surely there is abundant evidence of asymmetry in all regions of tumour growth, and as to the presence of what has been called a rarefying ostitis, a large number of the tumours of bone show the same, and did they not, I doubt whether its presence in this disease is sufficient for the conclusion that the disease is inflammatory, any more than the concurrence of atrophy of the muscles in pseudo-hypertrophic paralysis is sufficient to allow of the conclusion that that disease is a simple muscular atrophy.

I will only add that the disease osteitis deformans seems to me to present on the whole an obstacle to the recent doctrine that cancer, to use it as a very general term, is induced by some extraneous germ. At any rate this much may be said of it, that in *it*, and

its more usual outcome of tumour of some kind, we have an equally probable hypothesis that cancers are, through the line of hypertrophy and inflammation, but a misguided process of evolution, and I cannot but think that there is still room for a purely cellular theory of cancer. I would even put it stronger, and say that instead of seeing any strangeness in the occasional occurrence of a cancer, knowing what we do know of the physiology of cell life, it would be stranger still were something of the nature of cancer wanting in pathology. *March 20th*, 1888.

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16. *A case of osteitis deformans.* (*Card specimen.*)

By John R. Lunn.

The patient from whom these specimens (calvarium, clavicle, and lower jaw) were removed was a man, aged 68, subject to osteitis deformans, whose case was fully described in the Clinical Society's 'Transactions,' vol. xviii, pp. 274, 275. A year before he died he developed symptoms of bulbar paralysis with extreme wasting of his tongue, dysphagia, and some loss of taste; the lower jaw protruded three quarters of an inch beyond the upper.

Post-mortem notes.—Body is much emaciated. The femur on each side is curved outwards; there are six inches between knees when the feet are touching.

Skull: The dura mater is firmly adherent to the skull cap, which is enormously thickened, and weighs 1 lb. 11 oz. There are bosses on the orbital plates.

Brain weighs 2 lbs. 14 oz.; normal, except frontal lobes rather atrophied. Arteries rather atheromatous.

Lower jaw is much thickened and protruding. Tongue is very wasted.

Eyes: Double ectropion.

Thenar and interossei muscles much wasted; also wasting o muscles of thighs.

Femur was found to be brittle and could be easily cut through.

Spinal column: No displacement of vertebræ; marked curvature; spinous processes much thickened.

Spinal cord: Soft in cervical region; almost pulpy opposite first and second dorsal and seventh cervical vertebræ.

Right lung: Weight 1 lb. 8 oz.; hypostatic congestion.

Left lung: Same as right.

Heart: Weight 14 oz.; no clots; mitral valve thickened; left ventricle hypertrophied.

Aorta: Transverse patches of calcareous substance on anterior surface.

Other organs healthy. *May 15th*, 1888.

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17. *Caries without suppuration.*

By Frederic S. Eve.

I desire briefly to call the attention of the Society to some specimens of extensive caries without suppuration which seemed to me unusual. A familiar example of dry caries is that occurring on the articular surfaces of the bones in osteo-arthritis. It is also common in syphilis, especially on the skull. Here interstitial destruction of the bone takes place by the formation of circular patches of gummatous material in the cancellous spaces. In tuberculosis of bone, softening and suppuration are decidedly the rule, but I am under the impression that dry caries is not exceedingly rare even in the later stages of this disease. Clinically, many cases of extensive angular curvature are unaccompanied by external abscess, but probably in the majority of these the abscess does not make its way to the surface, and finally dries up. I may refer to this specimen (Fb. 54) from the London Hospital as an example of extensive superficial caries of the vertebræ without any evidence of suppuration, although there is a little caseous detritus about the eroded surface. The specimen is thus described in the Catalogue:

"Parts of the bodies of two adjacent vertebræ. The intervertebral substance has been destroyed, and a very considerable angular projection has been produced. In the bodies of both the vertebræ involved there are isolated cavities containing a structure not unlike crude tubercle. The specimen was removed from a man, aged 39, whose leg Mr. Luke had amputated on account of disease of the tarsal bones and ankle-joint. Two years previously an

ssue had been made in his back on accou it of the disease of the spine, and this was believed to have been arrested. A quantity of caseous deposit was found at the *post-mortem* around the seat of the disease, and a small fragment of dead bone was detached. From the notes it seems clear that no abscess had ever burst externally, and that no collection of fluid pus was found at the *post-mortem*."

I am able to exhibit another example from the Museum of the Royal College of Surgeons, Specimen No. 2065. It shows a vertical antero-posterior section of the spine in the dorso-lumbar region, with extensive caries of the adjacent surfaces of the last dorsal and first lumbar vertebræ, and destruction of the intervertebral substances. Towards the anterior part of the spine is a cavity filled with two small sequestra of cancellous tissue, and a little caseous material. Behind this is a firmer material composed of granulation tissue containing a few particles of bone. I have examined the specimen out of the bottle, and am unable to find the least trace of suppuration.[1]

Theoretically, suppuration in tuberculosis of the bone might be expected to be the exception rather than the rule. Visceral tuberculosis is not generally accompanied with suppuration unless the diseased part communicates directly or indirectly with one of the orifices of the body. Further, we should not expect the tubercle bacillus to have a pyogenic action unless accompanied by the micrococcus of suppuration. In fact, it often is thus accompanied. Suppuration is probably more common in bone than in internal organs, owing to its comparatively low vitality. Permit me now to draw your attention to two specimens of caries without suppuration which seem to me to possess exceptional characters. This specimen was presented to the Museum of Surgeons by Mr. Jonathan Hutchinson in 1882. I have described it in the Pathological Catalogue, No. 1284 A. It comprises a section of the os calcis. The whole of the cancellous tissue of the bone has been removed, and its place is occupied by soft granulation tissue containing a few spicula of bone. It is surrounded by a wall of slightly thickened bone. In the recent state pus could be detected neither in the

[1] Mr. Hutchinson has recorded a remarkable specimen of caries of the articular surfaces of a knee-joint without suppuration ('Trans. Path. Soc.,' vol. xxxviii, p. 312, 1887). Since writing this communication I have examined the articular end of the femur and found typical tuberculosis.

bone nor in the surrounding soft parts, and no sinuses were found. The adjacent parts were healthy. The granulation tissue showed no caseous deposits or patches of softening. Microscopically there was no evidence of tubercle. And in the soft connective tissue organisation and ossification appeared to be commencing. The specimen was removed from a delicate woman aged 50. Considerable swelling about the ankle had existed for many months, and she had suffered with aching pain in the heel. The patient made a good recovery, and as far as could be ascertained had no return of the disease. Mr. Hutchinson thinks that there was an indefinite family history of tuberculosis.

The second specimen was presented to the Museum by Mr. Buchanan, of Glasgow (see Catalogue, No. 1284 B) :—" It also is half of an os calcis divided vertically. Beneath the epiphysial cartilage of the tuberosity a band of cancellous tissue of considerable thickness has been destroyed and replaced by a layer of soft granulation tissue. The remaining cancellous tissue is rendered extremely hard and dense by new bone deposited in the cancellous spaces. Suppuration had not taken place. The os calcis was excised. From a girl aged 10, who had suffered with slight pain and swelling of the heel." The appearance and circumscribed character of the lesion, and the abundant evidence of repair in the remainder of the bone, are strong evidence against tuberculosis in this case, but I had not an opportunity of making a microscopic examination. The first case is much the more remarkable from the extreme destruction of the bone without suppuration. The view that the disease was not tubercular is supported by the absence of caseous material or of tubercle microscopically, and the signs of commencing repair. Such a case as this must not be confounded with the common examples of rarefactive ostitis, either as the early stage of simple inflammation or of tuberculosis of bone. Of these the former, as a rule, soon terminate in resolution or sclerosis, the latter in softening and suppuration. Bacteriology may also furnish us with the key to these cases of destructive disease of bone without suppuration, if we suppose that an inflammation has been excited by some trauma, and that pyogenic micrococci have failed to gain access to the inflammatory focus.

I may also draw attention to a specimen of widely destructive dry caries of the knee-joint previously exhibited by Dr. Lediard ('Transactions,' vol. xxxiv, 1883, p. 229), and now preserved in the

Royal College of Surgeons Museum. Dr. Lediard has kindly sent me further details of the case, but no explanation of the peculiar character of the disease has come to light. *February 7th*, 1888.

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18. *Part of an ancient Egyptian skull affected with osteo-porosis.* (*Card specimen.*)

By Frederic S. Eve.

A portion of a parietal bone affected with osteo-porosis. It was found in an Egyptian tomb, at Tell Nebesheh, of the period Saite to Ptolemaic. The bone is much thickened, and composed throughout of finely porous cancellous tissue. Its surface is rough and marked by numerous arborescent grooves and minute apertures. On its inner surface the canals for blood-vessels are deepened. The condition of the bone does not correspond altogether with the appearances of osteitis deformans.

The specimen was presented to the Royal College of Surgeons Museum by the Egyptian Exploration Society.

October 18*th*, 1887.

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19. *Case of myxoma of cranium.*

By Ernest H. Jacob, M.D.

[With Plate XVI, fig. 2.]

J. T—, aged 55, was admitted to the Leeds Infirmary on July 19th, 1887, under the care of Dr. Eddison. He was in a comatose condition, and died a few hours after admission. The autopsy showed the cause of death to be uræmia from granular kidneys, but the cranial condition was remarkable, though apparently it had given rise to no peculiar symptoms.

Externally there was considerable bulging over the left parietal and occipital region, especially at the posterior aspect, beginning

about an inch to the right of the middle line, extending backwards almost to the base of the skull. The surface was rough, hard, and nodular, except for a space about three or four inches immediately above the squamous part of the temporal, where the skull was soft and perforated by a number of small holes. Something of the same condition was also seen at the external angular process of the frontal on the same side.

A section made transversely from side to side through the most prominent part of the projecting mass showed it to be formed by a soft growth of white colour growing from the diploë and distending the tables (Pl. XVI, fig. 2). The greatest thickness was an inch and a quarter, the outer table being here about one sixteenth of an inch thick, and the inner rather less. The growth extended over the whole of the parietal and a small part of the frontal region as described above, but there was general thickening of the bone in the occipital region, and for about an inch from the upper part of the squamous. The bone of the whole of the upper part of the cranium was unusually hard. There was very little encroachment on the cranial cavity, the upper table having yielded to the pressure of the growth. Microscopically the growth appeared to consist of myxomatous tissue interspersed with spicules of bone.

October 18*th*, 1887.

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20. *A parosteal lipoma, or congenital fatty tumour, connected with the periosteum of the femur.*

By D'Arcy Power, M.B.

The specimen which I exhibit to you this evening presents many points of interest. It is an example of the comparatively rare form of the congenital fatty tumour which grows from, or has developed an intimate relation with, the periosteum. To this form of tumour the term parosteal lipoma has been applied. The tumour was removed from the thigh of a boy aged 9 years, who was admitted into St. Bartholomew's Hospital under the care of Mr. Thomas Smith. The following notes were taken at the time of his admission:—"He has a soft, painless, and elastic swelling extending

over the upper third of the left thigh, and along its outer aspect. The skin is freely movable over the tumour, but the superficial veins in this region are somewhat distended. The left thigh measures seventeen inches in circumference at its thickest part, whilst the right thigh at the corresponding point only measures fourteen and a half inches." The tumour was thought to contain pus, and was punctured with an aspirating needle; a small quantity of blood was alone obtained. An incision was subsequently made over the swelling, and the tumour was found to be fatty in nature, and to be firmly attached to the periosteum of the femur by a broad base which commenced immediately below the lesser trochanter. The line of attachment was so firm that considerable difficulty was experienced in removing the tumour. The patient made a good recovery except for a trifling secondary hæmorrhage which was readily controlled.

The tumour is large and irregular, weighing 15 oz., and measuring six inches by five. It is divided into several lobes which are held together by areolar tissue, but it is only partially encapsuled. Many pieces of muscular tissue are attached to the capsule in different parts, but I believe that their presence is purely accidental, and that they are not in any way to be taken as indications that the tumour grew from the deeper intermuscular planes of connective tissue, only secondarily becoming attached to the periosteum. The growth is extremely firm, and consists of fat held together by trabeculæ of dense fibrous tissue. Microscopical examination confirms the naked-eye appearances, for the sections show collections of fat-cells, separated by thick bundles of connective tissue. There is no muscular fibre in the growth so far as I have examined it, nor does it appear to be undergoing any degenerative changes.

Parosteal lipomata are rare, and indeed the author of the article on tumours of bone in Ashhurst's 'Encyclopædia of Surgery'[1] only alludes to three cases of the kind, and states that "upon these three observations rests the belief in this variety of neoplasm." I imagine, however, that they are not so rare as this statement would imply, for accounts of similar tumours have been recorded, though I do not find them to be very numerous. Of these the most interesting to the Society are those alluded to by our President in his 'Lectures on Surgical Pathology';[2] and those exhibited by Mr.

[1] Vol. vi, p. 980.

[2] Ed. i, Lond., 1853, vol. ii, p. 98.

homas Smith in 1866 and 1868;[1] and by Mr. John Wood[2] in 1875. Mr. Butlin,[3] too, has written an instructive paper upon growths of is nature.

The points of especial interest about the growth are, that it is a congenital fatty tumour, and this in itself is extremely rare. Secondly, it is attached to the periosteum, a very unusual feature. Upon this point Mr. Butlin says,[4] "It seems very probable that such growths commence as fibrous or fibro-cellular tumours, and that the formation of fat is a later change, not very late perhaps, but occurring soon after the first appearance of the tumour, and proceeding almost collaterally with its growth. The large quantity of fibrous tissue contained in most of these tumours, the ill-developed nature of the adipose tissue as shown by the microscope, and the strange fact that nearly half the cases collected are stated to have been attached to bone or periosteum, sometimes immediately, and sometimes by a fibrous pedicle, whilst several of them are stated to have actually arisen from bone, have led me to this belief. A less likely bed than the periosteum for the origin of a fatty tumour can scarcely be imagined, whereas it is one of the ordinary seats of origin of a fibroma." It appears to me, however, that there is no special reason why lipomata should not spring from the periosteum, for they are connective-tissue growths commencing as localised proliferations of cells which rapidly become infiltrated with fat.

The last point of interest which I shall touch upon in connection with the tumour is the fact that the capsule is deficient over the greater part of its circumference. The shaggy appearance of the diffuse portion of the growth contrasts well with the smooth outline of that which is bounded by a well-defined fibro-cellular coat.

The specimen is preserved in the museum of St. Bartholomew's Hospital, Series i, No. 436 (*a*). *February 21st*, 1888.

[1] 'Trans. of the Path. Soc.,' vol. xvii, p. 286, and vol. xix, p. 344.

[2] Ib., vol. xxvi, p. 190.

[3] 'St. Bartholomew's Hospital Reports,' vol. xiii (1877), pp. 179—185.

[4] Op. cit., p. 184.

Fig 2.

DESCRIPTION OF PLATE XVIII.

Figs. 1 and 2 illustrate Mr. Eve's case of Central Fibrosarcoma of Tibia with Cystic Degeneration. (Page 273.)

Fig. 1.—Section showing degeneration, apparently mucoid, of a portion of the tumour, this change being probably the precursor of cyst formation. Zeiss D, oc. I.

Fig. 2.—Section showing the microscopic appearances of the bulk of the tumour. It exhibits wavy bands of well-developed fibrous tissue studded with a few nuclei.

Fig. 3 illustrates Mr. Eve's case of Tuberculosis of the Skin of the Hand. (Page 369.)

The section shows a miliary tubercle containing three giant-cells. × 320 diam.

Fig. 4.—Drawing of a Rabbit's Head after Inoculation with Lupus, exhibited by Mr. Eve. (Page 365.)

Near the base of the right ear is a large dry ulcer, which followed the subcutaneous insertion of fragments from a case of lupus vulgaris. Nearer the tip of the ear is a similar but much smaller ulcer which developed at a distance from the seat of primary inoculation. There is also superficial ulceration of the posterior margin of the ear. On the left ear is a cold abscess the result of inoculation of pus from the ulcer on the right ear.

21. *Specimen of central fibro-sarcoma expanding tibia, accompanied by extreme cystic degeneration; with remarks on the relation of injury and inflammation to the production of sarcoma of the bones.*

By Frederic S. Eve.

[With Plate XVIII, figs. 1 and 2.]

The tibia was greatly enlarged and fusiform. On section, its walls were found to be evenly expanded by the growth of a central sarcoma, which occupied its whole length. In the upper half of the section the tumour had completely undergone cystic degeneration, the cysts being of large size, with smooth walls, and separated from each other only by thin septa. In the lower half it was composed of homogeneous or faintly fibrillated substance enclosing only a few cysts. The remains of the tibia formed a smooth, thin, and almost complete wall around the tumour. The expansion of the bone extended up to the articular ends, which were intact. The outer wall, after maceration, showed a trabeculation or basket-work. The cysts contained a yellowish fluid, very rich in cholesterine, which formed a thick coating of silvery particles.

Under the microscope the firmer parts of the tumour were composed of broad wavy bands of well-formed fibrous tissue, studded with a few flattened nuclei (Pl. XVIII, fig. 2). From this transitions passed to a lowly-organised tissue consisting of round nuclei separated by a scanty homogeneous, or imperfectly fibrillated stroma. In some sections a mucoid degeneration of the tissues was observed, leading to softening and the formation of spaces (fig. 1); it was doubtless by this means that the cysts originated.

The specimen was removed, by amputation at the knee-joint, from a man aged 24. Ten years previously he sustained a fracture of the tibia a little below the middle. "It had ached more or less ever since, especially during changes in the weather. A small lump always remained in the neighbourhood of the break; but nine months ago the leg began to enlarge generally, and has continued to grow and to pain him since. He continued at his work until his admission into hospital." The above details are

taken from a letter by Mr. W. T. Clegg, of Liverpool, who presented the specimen to the Museum of the Royal College of Surgeons, where it is preserved.

Remarks.—The specimen above described offers several points of interest. In the first place, it is unusual for a central tumour of bone to be microscopically so highly organized; they are usually round-celled or myeloid sarcomata. In fact, with the exception of some tumours of the lower jaw and a subperiosteal tumour in a dog in St. Bartholomew's Hospital Museum, I have never met with a tumour of bone so highly organized; that is, in which the cellular or embryonic elements had to so large an extent undergone development into fibrous tissue. Again, such an extensive cyst formation in a sarcomatous tumour of bone is, in my experience, unparalleled, and the mode of formation of the cyst with the occurrence of cholesterine are of some pathological interest.

Several observers, myself included, have drawn attention to the etiological relation between injuries and inflammations of bone and tumour formations. As already stated, a fracture of the limb occurred, in the case related, nine or ten years before the appearance of the tumour; and from the history of persistent pain and swelling at the seat of fracture it is probable that a condition of chronic inflammation remained.

Considering the highly organized character of the tumour it does not seem too wide a stretch of the imagination to compare it to overgrown callus-tissue, which, instead of ossifying, has gone on continuously growing.

In some respects it presents some affinities with a remarkable specimen[1] exhibited at this Society by Mr. Bilton Pollard ('Transactions,' vol. xxxvi, 1885, p. 388), in which a growth of uncertain nature, but resembling microscopically callus-tissue, occurred as a fusiform swelling involving the whole thickness of the tibia of a child aged 5 years. A year before the appearance of the swelling the child sustained a severe injury to the leg, but it was uncertain if it had been broken. The specimen was submitted for examination to a full meeting of the Morbid Growths Committee of the Pathological Society, and the members could not come to a unanimous opinion, one half believing the growth to be an inflammatory new formation allied to the fibrous matrix of callus, the other half preferring to call it a sarcoma.

[1] Preserved in Royal College of Surgeons Museum, No. 1241 D.

I might instance many other tumours of bone, which were apparently excited by a blow, fracture, or other injury, but will only mention three, in some respects remarkable. They are both in the museum of St. Bartholomew's Hospital. One (No. 454), like the specimen forming the subject of this paper, is a fibro-sarcoma, and springs from the front and inner surface of the tibia. The patient, a woman aged 32, sustained a fracture of the middle of the leg, and shortly afterwards the tumour appeared. Three years later she again fractured the same bone lower down, and the line of fracture is still distinguishable. The tumour grew very slowly, for it was first noticed eight years before the removal of the limb by amputation.

No. 420 is an osteo-chondroma of a femur which followed a fracture.

Nos. 419 and 420 show sections of a femur with an enchondroma, which is said to have followed a fracture. The next two specimens contain sections of the humerus of the same person. The bone is affected with a similar tumour, and has been fractured.

Since cartilage does not form after fractures in adult bones it is of course open to question whether the last-mentioned tumours had not originated in small islands of embryonic cartilage left unabsorbed, and that these by their growth had weakened the bone and caused the fracture.

With regard to inflammation, I may refer to another remarkable specimen (No. 450) of spindle-celled sarcoma surrounding the lower third of a femur. The corresponding portion of the medullary canal is dilated, and lying within it is a sequestrum of cancellous tissue. That this was a case of quiet necrosis preceding the tumour-formation is rendered most probable from the circumstance that the osseous tissue of the shafts of bones surrounded by tumours is almost invariably sclerosed, and the medullary canal obliterated.

The relations of injury and inflammation as exciting causes of tumours of bone are too evident to be doubted, but whether the inflammatory lesion alone is sufficient to induce the morbid growth, or whether the increased blood supply stimulates to activity some virus, or some mass of embryonic tissue, cannot yet be determined. My own bias is in favour of the view that a long-continued or repeated inflammatory condition is sufficient, when associated with

certain inherited or acquired tendencies in the tissues. That as in certain individuals an overgrowth of fibrous tissue or keloid occurs at the seat of scars or in the lobes of the ears from the irritation of ear-rings, so in others a long-continued, chronic, inflammatory condition may produce tissues with inordinate power of growth and defective organization, namely, a tumour. It is to me quite conceivable that a cell may become so bloated by over-feeding—or a too abundant supply of plasma—as to be capable of indefinite multiplication, its progeny living in unusual localities and destroying less highly-energized tissues.

Such a change of habit and character can be accomplished in some micro-organisms—themselves merely cells—by varying the culture fluid, or by passing them through the bodies of animals more susceptible to their attacks. Why may not the leucocytes be similarly modified? For, of all the cellular constituents of the body, they appear to be the most independent of the other tissues, and they probably constitute the chief source of most of the sarcomata. *November 15th*, 1887.

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22. *Necrosis of inferior maxilla associated with sarcoma.* (*Card specimen.*)

By W. H. Battle.

W. B—, aged 20, a farm labourer, was admitted, under the care of Mr. Sydney Jones, on October 14th, 1887, and left December 4th, 1887.

He suffered much from toothache and went to a dentist about a year before admission. The second molar on the left side was broken during attempts at extraction. Soon afterwards the jaw commenced to swell. Subsequently an abscess broke in the mouth. Seven weeks ago he became so ill that he was obliged to give up work. Since then abscesses have broken below the ramus of the jaw, and in the supra-clavicular region, the discharge being very offensive.

On admission, a sinus about midway between the symphysis and angle of jaw on the left side. Above the clavicle and extending

from it up into the neck is a dark inflamed area, and in it are four or five sinuses discharging pus freely. There is also much swelling with tenderness about the lower jaw on the left side. Patient rather anæmic, otherwise in good health.

Incisions were required to relieve the condition of the neck, and on October 26th Mr. Pitts examined the jaw, which was found completely hollowed out as far as the alveolus. Some of the products removed by the sharp spoon were examined microscopically and found to be characteristic of spindle-celled sarcoma.

At the request of Mr. Sydney Jones, on October 26th I removed the whole of the left half of the lower jaw. On examination, it was found to be enlarged laterally from the second bicuspid to the angle. The greatest width of the enlargement was opposite the second molar, where it measured one and a half inches, the increase in size being most marked on the outer surface. On making a section through it, there is seen on the superior inferior and external surfaces, a thin layer of bone (about one eighth of an inch thick), while on the inner side there is a much thicker (a quarter to a half an inch) layer of bone. Between them is enclosed a tumour somewhat horse-shoe shaped on section, measuring about an inch across. The concavity of the horse-shoe is formed by an abscess cavity in which was found a sequestrum. The tumour itself feels tough and fairly hard. A probe is easily passed into this cavity from the alveolus of the second molar.

Examination of the growth after the removal of the jaw, reconfirmed the diagnosis of spindle-celled sarcoma. The patient was quite well when he left the hospital, and has continued well since, the jaw being replaced by a dense band of cicatricial tissue.

May 15*th*, 1888.

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23. *Melanotic sarcoma in the head of the tibia.* (*Card specimen.*)

By C. Stonham.

The tumour has fungated through an opening in the soft parts. The growth consists of separate centres, the highest and most posterior being separated from the others by bone. There is

a good deal of bone in the substance of the tumours, which have infiltrated the cancellous tissue.

The patient, a woman aged 39, had had a primary growth removed some years previously from the right supra-clavicular region; this was of small size.

The growth in the tibia was first noticed in November, 1886. in January, 1887, it was scraped away, but reappearing, Mr. Heath successfully amputated through the condyles in March, 1887. On leaving the hospital the patient had numerous recurrent growths in the skin.

University College Museum, No. 676A. *May 15th*, 1888.

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24. *Knee-joint showing the changes occurring in rheumatoid arthritis.* (*Card specimen.*)

By C. Stonham.

The cartilage has been extensively destroyed; that which remains is thick and roughened, and resembles at first sight a deposit of urate of soda. The denuded bone is eburnated. At the margins there are bosses of new bone covered with cartilage. This is especially well seen on the femur. The synovial membrane is very slightly villous in places, the processes being very delicate. The outer margin of the tibia is raised by a bony outgrowth covered with cartilage, so that the articular surface is considerably deepened.

The specimen was removed from a man, aged 68, who was under the care of Mr. Beck at University College Hospital. He died from malignant disease of the œsophagus with recurrences in the bones. He had suffered from rheumatism for many years. The other knee-joint and both elbows were also affected, but the disease was very early. The shoulders were in an advanced stage. Both hip-joints were also diseased considerably.

The specimen is preserved in the University College Museum.

February 7th, 1888.

25. *A knee-joint showing the changes occurring in rheumatoid arthritis.* (*Card specimen.*)

By C. Stonham.

The articular surfaces are much increased in area owing to the overgrowth which has taken place at the margins of the cartilage. The central portion of the cartilage does not show a proportionate amount of disease; it is not markedly roughened nor has it been worn away except in a small area on the femur. The patella is uniformly enlarged, being almost double the normal size; the peripheral outgrowth of cartilages is very marked. The semilunar cartilages are much atrophied. The synovial membrane exhibits very beautifully the villous condition met with in this disease. These processes vary a good deal in size, some of them (as on the outer condyle) being very delicate; others (inner condyle) are much coarser.

The patient was under the care of Dr. Ringer in University College Hospital, June, 1887. The other joint was in a similar condition. The specimen is in the Museum of University College.

February 7th, 1888.

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26. *A knee-joint showing the changes occurring in gout and an early stage of rheumatoid arthritis.* (*Card specimen.*)

By C. Stonham.

The cartilage is for the most part smooth, but here and there is pitted irregularly, especially where the urate of soda is deposited; this is well seen on the patella. Urate of soda is also deposited in the semilunar cartilages. The margin of the cartilage encrusting the femur has an appearance of thickening as in an early case of rheumatoid arthritis. The synovial membrane is in a villous condition, especially above the condyles. The villi are club-shaped and vary in length up to three-quarters of an inch; many are very slender and short.

The patient was under the care of Dr. Ringer, in University College Hospital, in July, 1887. He was aged 53, and had suffered from rheumatism for ten years, and gout two years. He died from the effects of valvular disease of the heart, death occurring suddenly from syncope.

(Specimen preserved in the Museum of University College.)

February 7th, 1888.

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27. *Osseous ankylosis of the metatarso-phalangeal articulation after gouty arthritis.* (*Card specimen.*)

By Samuel G. Shattock.

Section reveals a perfect continuity of healthy cancellated tissue between the bones concerned, no uratic deposit being present in the osseous substance.

There is but little external deformity, and the ankylosis has occurred in the extended position. In the tissue immediately around the ankylosed joint there are some flattened deposits of urate of soda. There was extensive gouty disease of, and uratic deposit around, many of the larger joints of the body.

March 20th, 1888.

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28. *Uratic deposit in the head of the first phalanx of one of the fingers.* (*Card specimen.*)

By Samuel G. Shattock.

The cancellated tissue of the phalanx is filled for a distance of a centimetre with urate of soda. The articular cartilage is wanting; that of the opposed phalanx is infiltrated with urate. Large tophi surround the diseased joint beneath the extensor and the flexor tendons. *March 20th*, 1888.

29. *Rare forms of loose bodies from the knee-joints.* (*Card specimens.*)

By Anthony A. Bowlby.

Case 1.—Two portions of cartilage removed from the knee-joints of a lad aged 18 years. They were almost exactly alike in form and size, each resembling such a piece of cartilage as might be obtained by removing that which covers the inferior surface of one of the condyles of a femur, and each, as such a piece would be, is smooth and polished on its convex, and rough on its concave surface.

Microscopical examination shows that the specimens present all the appearances of articular cartilage, although at their deeper parts there is an abnormal formation of fibrous tissue. The patient gave no history of injury, and on his admission to the hospital complained of one knee only. From this point one of the pieces of loose cartilage was removed with a good result, and for a year nothing more was heard of the lad. At this time he returned with symptoms of a loose body in the opposite knee, and from this articulation the other loose body was then removed with a similar good result.

(See St. Bartholomew's Hospital Museum, No. 721.)

Case 2.—*Multiple loose cartilages from the knee.*—Four hundred and fifteen loose cartilages removed by operation from the knee-joint. They are all of irregular shape, with nodulated, uneven surfaces, and vary in size from a swan shot to an almond, by far the larger number being midway between these two sizes; the largest and the smallest are suspended at the top of the bottle. They are none of them pedunculated, and were not attached to the synovial membrane. Three of them may be seen to be very nearly divided into two separate portions, which yet remain united by a thin fibrous thread. A cut section is smooth, homogeneous, and glistening; a microscopic examination showed that they consisted of hyaline cartilage.

From a man, aged 31, who had had four attacks of rheumatic fever, whilst for six years the knee-joint had been painful and swollen. At the time of the operation the synovial membrane was seen to be rough and vascular like granulation tissue.

(See St. Bartholomew's Hospital Museum, No. 726 A.)

Case 3.—*Loose body from knee-joint formed by blood extravasation in the synovial membrane.*—The patient from whom this body was removed was a healthy man of 24. Whilst playing football he severely wrenched his knee in the following manner. He ran to kick the ball, but missing his aim, his leg was violently extended at the knee-joint and the limb was at once rendered useless by the severe pain which was caused. The patient described a sensation as of something being torn in the joint. The injury was followed by acute synovitis, and then by chronic effusion of synovial fluid; in four months' time, when the effusion subsided, a loose body was distinctly felt.

This body was removed by Mr. Langton by direct incision, and was found to be attached by a pedicle to the synovial membrane. It consists of two folds of synovial membrane enclosing between them some partially decolourised blood-clot. The loose body appears to have resulted from hæmorrhage into a torn synovial fringe.

(See St. Bartholomew's Hospital Museum, No. 716 B.)

Case 4.—*Loose body from knee-joint.*—A flattened, pedunculated ragged mass of synovial membrane removed by operation from the knee-joint of a man, aged 32, who had suffered from symptoms of "loose cartilage" for several years.

A microscopical examination shows that the synovial membrane is infiltrated with inflammatory exudation and fibrous tissue.

The loose body appears to have originated in a tear of the synovial membrane, the torn piece being subsequently dragged upon in the movements of the articulation.

(See St. Bartholomew's Hospital Museum, No. 716 A.)

Case 5. *Partially detached semilunar cartilage.*—This specimen was removed from a healthy man, aged 26, who had frequently wrenched his knee whilst playing football and cricket, and had latterly suffered from all the symptoms of "loose cartilage." On admission into St. Bartholomew's Hospital a loose body was readily felt in the left knee-joint; it was placed on the outer side, and its range of movement was somewhat limited.

The loose body was removed by Mr. Langton, who found that the external semilunar cartilage had been torn from its posterior

attachment, and was further detached laterally for one half or more of its length. The torn piece of cartilage was severed from that portion which remained attached, and was removed.

The five preceding cases are examples of the more rare forms of loose bodies in joints. The first specimen appears undoubtedly to afford an example of the detachment of a piece of articular cartilage, an accident which is generally supposed to result from a direct blow rather than from a strain or wrench. But, although this explanation may apply to other cases of similar loose bodies, it can scarcely be held to account for the specimens here exhibited. There was no history of any injury, and the fact that portions of cartilage of almost exactly similar shape and size were removed from each knee-joint, renders the theory of injury quite untenable. In the absence of injury, however, it becomes exceedingly difficult to assign a cause for this phenomenon. I am, nevertheless, disposed to attribute the separation of the cartilage to some such cause as scarlatina or typhoid fever. It is well known that after such affections certain localised portions of bone are liable to necrose, and other tissues, such as the veins and the joints, are liable to attacks of inflammation. It appears to me that some such general cause is more likely to account for the symmetry of the lesions than is any local injury. Unfortunately no inquiry was made of the patient as to any previous illness, and the theory I have advanced is therefore unsupported by any positive evidence.

Case 2 is remarkable rather for the number than for the character of the loose bodies, yet it is noteworthy that all the bodies are of almost exactly the same size, that none are calcified, and that only four have any pedicles.

Case 3 affords an example of a hitherto undescribed mode of formation of loose bodies, although I have already alluded to this specimen and figured it in my recent 'Surgical Pathology.' It is true that the formation of loose bodies has been attributed to hæmorrhage into the cavities of the joints, but I am not aware of any example of a loose body actually formed by hæmorrhage into the synovial membrane itself.

It is quite possible that the specimen in Case 4 may have originated, as did Case 3, by a tear followed by hæmorrhage into the synovial membrane; but if this were true it is evident that the loose body has subsequently been increased in size by the

exudation of inflammatory products and the formation of fibrous tissue.

The last specimen is in some respects the most interesting, for although separation of the marginal attachment of the semilunar cartilages has already been described, this specimen shows that the very strong posterior attachment may be torn through, and may allow a very free movement of the detached cartilage. It is also noteworthy that, although the knee had several times been wrenched, there was no history of such great violence at any time as to incapacitate this patient for walking. *April 17th*, 1888.

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30. *Symmetrical loose bodies from two knee-joints.* (*Card specimen.*)

By H. H. Clutton.

The two flat cartilaginous bodies which are mounted together in the accompanying bottle, were removed successfully from the knee-joints of a lad who at the time of the first operation was fifteen years of age.

The first was removed in May, 1885, from the left knee, the patient denying any history of injury, and having suffered from symptoms of "loose cartilage" for seven weeks.

The second was removed in September, 1887, from the right knee, the patient having suffered from symptoms for two months, and stating that two years before he had strained the right knee by a fall.

The two bodies are curiously like one another, both being flat and of much the same shape and size. The first, and slightly smaller of the two, is nearly an inch in its longest diameter, and half an inch in breadth, whilst in thickness it measures about one eighth of an inch.

The second is not quite so regular in outline and shows a slight increase over the first body in all its measurements.

Microscopically, there is no evidence to show that they came from the articular cartilage. Although mainly composed of cartilage the cells are distributed irregularly. Calcification has taken

place in some parts but there is no bone to be found in the sections which were kindly made for me by Mr. Shattock.

April 17th, 1888.

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31. *A rare form of pedunculated body in the knee-joint.*

By Samuel G. Shattock.

Had it not been that Mr. Bowlby has alluded to a similar though considerably smaller specimen in the museum of St. Bartholomew's ('Surgical Pathology,' 1887, p. 325), the present specimen might, I believe, have been designated as an unrecorded variety of pedunculated body. No such form is, so far as I can ascertain, mentioned by surgical writers.

The knee-joint was handed over to me by Dr. Reid from a dissecting-room subject.

The bones and other structures forming the joint were quite healthy, except for some fibrous metaplasia of the articular cartilage of the patella.

Projecting into the joint, on the right side and immediately below the patella, is a discoidal body 4 cm. in the transverse diameter, 3·2 cm. in the vertical, ·7 cm. in thickness. It is invested with a thin capsule of connective tissue continued into a falciform synovial fold on the right side, which fold is lost by the adjacent border of the patella in the general synovial membrane.

The body lies immediately below the patella, passing a little way beyond the middle line to the left side; by its upper part it rests on the lower triangular non-articular division of the posterior surface of the bone, its upper border being accurately adapted to the lower margin of the articular surface. At first sight it appeared to be simply a local overgrowth of the fat which is naturally present in the synovial fold of the alar ligament.

There is an excellent example of such a lipoma growing in this situation in the museum of St. Bartholomew's (No. 716), and Mr. Barwell states ('International Encyclopædia of Surgery,' vol. iv, "Diseases of Joints") that he has removed an intra-articular

lipoma from each side of the patella. A closer inspection, however, discloses through its surface blotches of the dull orange red, characteristic of hæmorrhagic extravasations in which secondary changes have occurred, whilst an incision shows the pendulous body to be quite firm and compact in substance, without discernible structure, and of greyish-white colour marbled with rusty red.

Histology.—Vertical sections stained with hæmatoxylin show a faintly stained meshwork of a homogeneous or finely granular or nebulous substance, in the meshes of which are collections of reddish brown granular pigment. The meshwork is in some places coarse, with thick intercommunicating trabeculæ; in others the trabeculæ are finer, and arranged with some degree of parallelism. In the latter parts the structure of true fibrous tissue is closely simulated; the simulation is increased by the presence here and there of sparsely distributed corpuscles—the leucocytes of the blood originally shed, or cells of the delicate and lax connective tissue into which the extravasation has occurred. Such an appearance is, indeed, the nearest approach to self-organisation of which blood-clot is capable. As contrasted with true fibrous tissue, however, the substance of the trabeculæ shows no trace of striation, but is quite homogeneous; the trabeculæ freely intercommunicate, and they take a stain in varying degrees with hæmatoxylin, which the fibres of the true tissue never do. In the great bulk of the substance there are few, and in large tracks no visible corpuscles. The size of the coagulum and the tenuity of its connection with the surrounding tissues, practically place it beyond the pale of organisation.

Remarks.—This form of pedunculated body is clearly, therefore, due to old hæmorrhage into the alar ligament, arising, doubtless, from a contusion or similar injury, the result either of external violence or of injury sustained by the fold during some unusual movement of the joint. There is no evidence to warrant the belief that such a body as that under consideration could ever become organised, or rather replaced by the fibrous tissue, and such as constitutes the substance of some of the loose bodies met with in the joints. Nor must it be forgotten, in discussing the nature of loose bodies in joints, that one form *is* constituted by unorganised substance similar to that which constitutes the so-called melon-seed bodies met with in the other anatomical dispositions of synovial membranes, to wit, tendon sheaths and bursæ. There are very

few examples of such in museums. They are known, however, to occur, amongst other cases, in tubercular hydrops of the joints.

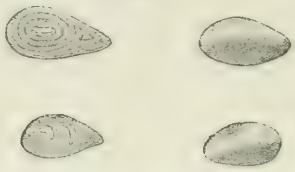
In one of the examples of intermuscular synovial cysts recorded by Mr. D'Arcy Power in the last volume of the Society's 'Transactions,' p. 341, it is noted that the cyst which occupied in part the position of a bursa between the semi-membranous and semi-tendinosus tendons, contained serous fluid in which floated a large number of melon-seed bodies. The cyst was in no communication with the knee-joint, but the synovial membrane of the knee presented microscopically all the appearances of tubercular disease; and I would suggest that the disease of the cyst was of a similar nature. The cyst is irregularly lined with lymph; the loose bodies are detached portions of the lining (1205 F. St. Bartholomew's). And sometimes such unorganised bodies are constituted in part by blood. There is a specimen of this kind in University College Museum (No. 943), which is thus described in the catalogue: "Several flattened bodies from a knee-joint, most of them approaching an oval form, the largest measuring fully half an inch in length; all of them consist of a firm, structureless, friable substance, of a pale brown colour; some are concentrically laminated. The bodies escaped with the pus from a diseased knee-joint when laid open; the limb was subsequently amputated by Mr. Liston."

Histologically, they consist, like the next specimen, of a homogeneous or hazy substance arranged in coarse trabeculæ, the peripheral and finer of which exhibit a concentric disposition; the meshes between the outer inter-communicating lamellæ hold a finely granular material, in which in some situations a segmentation is discernible, as though it were constituted by altered leucocytes. Granules of blood-pigment occur sparsely in the section.

The best example, however, is the following, placed amongst the series of loose bodies from joints, in the museum of St. Thomas's Hospital (D 105). The bodies in question number about fifteen, the largest is about one centimetre in its chief diameter. Some of them are regularly oval, others polyhedral or wedge-shaped. They have on section a very distinct lamination, quite obvious to the naked eye, and are of a reddish-brown colour. The laminated substance consists, microscopically, of homogeneous or slightly nebulous material, in coarse, irregularly-formed, concentrically disposed trabeculæ, the lamination being coarser and less evident towards the centre; scanty, deeply stained nuclei, spheroidal or

oval in shape, lie in the clefts of the substance towards the centre, and here and there a large granular plasma cell. The colouration is due to the presence of minute brownish red granules, in places

Woodcut 14.



Unorganised bodies from a joint.

in small shapeless groups, and more particularly abundant in the peripheral parts. The most interesting disclosure, however, is the existence of a distinct nucleus in these bodies of different structure from the main substance (see Woodcut 14). In some of them there are two nuclei; and in such, the succeeding lamination is at first in two systems, and then in one common to both, as are the strata seen when two or more urinary calculi come to be invested with a series of common laminæ, through means of which they are thus combined.

The nucleus, which is a well-defined isolable structure, contrasting in unstained specimens with the rest by its pure white colour, is composed of close wavy bundles of delicate connective tissue; and, like such tissue, it stains well with carmine, but is not stained in logwood preparations.

In this case, therefore, it would seem that the loose bodies have arisen by the detachment of the villous processes first described by Rainey as occurring on synovial membranes generally, and with these their nuclei correspond in microscopic characters, these villi having been before their detachment (and probably afterwards as well), the seats of laminated depositions from inflammatory effusion poured into the joint; and with this effusion there is evidence of admixture of red corpuscles in the pigment granules present in the laminated substance.

Such nuclei, however, do not occur in all, and here, as in the case of the melon-seed bodies of tendon-sheaths and bursæ, some such bodies have an independent origin or arise by depositions occurring on fragments of fibrin detached from the inner surface of the synovial membrane. That this is so in the cases referred to I have

been able to show in some instances by microscopic section; nor is it difficult in collections of such bodies to trace all gradations from those that are of a smoothly oval seed-like form down to shapeless shreds, and flakes of similarly unorganised substance. In other instances I have been able to demonstrate true fibrous tissue associated with the superposed unorganised substance.

In very rare cases it would appear (from a specimen in University College, No. 944) that such laminated bodies in the joints may become calcified. And they may be told from the calcified cartilaginous or other outgrowths met with in joints or bursæ, by their very compact and uniform composition, and by—what is pathognomonic—the lamination which remains evident in the calcified substance. They are, indeed, in such a condition closely like the phleboliths so common in the prostatic and ovarian plexuses; and if any loose bodies deserve the name, such might fairly be designated arthroliths. *April* 17*th*, 1888.

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32. *Dislocated internal semilunar cartilage.*

By H. A. Lediard, M.D.

Removed from a patient aged 19. The knee was first injured eighteen months prior to operation, when he was taking a long jump. Subsequently various injuries at football and cricket caused repeated attacks of synovitis, whilst a loose body was frequently found to project from the inner side of the joint.

On cutting down upon the supposed loose cartilage, it was found to be a loop of ligamentous appearance. On hooking the loop a free end appeared, but the other end was thick and fixed to near the spine of the tibia, and was cut away with scissors. The specimen, which measures three inches long by a quarter of an inch wide, is stout and fibrous towards its attached end, but thin and frayed out at the free end. It is believed to be the internal semilunar cartilage.

Microscopic examination shows a nucleated fibrous structure but no cartilage.

The patient has felt no inconvenience in the joint since.

April 17*th*, 1888.

33. *Enlarged bursa patellæ with endogenous growths.*

By H. A. Lediard, M.D.

This specimen was removed from the knee of a woman aged 25. It measured about four inches long by three broad, and had been growing for three and half years. There were two points of ulceration of the skin over it, from one of which straw-coloured fluid could be made to exude on pressure.

The bursa was excised, and on section was found to be lined with papillomatous growths, which were held together by fine threads of fibrine. These growths were easily detached from the wall of the bursa, and in colour were white.

The bursa itself was much thickened, and contained several ounces of clear straw-coloured fluid, but no pus.

Microscopic examination showed the growths to be without structure. This condition, so well described by Brodie, seems nearly related to those in which loose bodies are found in the knee-joint in large numbers. *April 17th*, 1888.

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34. *A case of "lipoma arborescens" (Müller).*

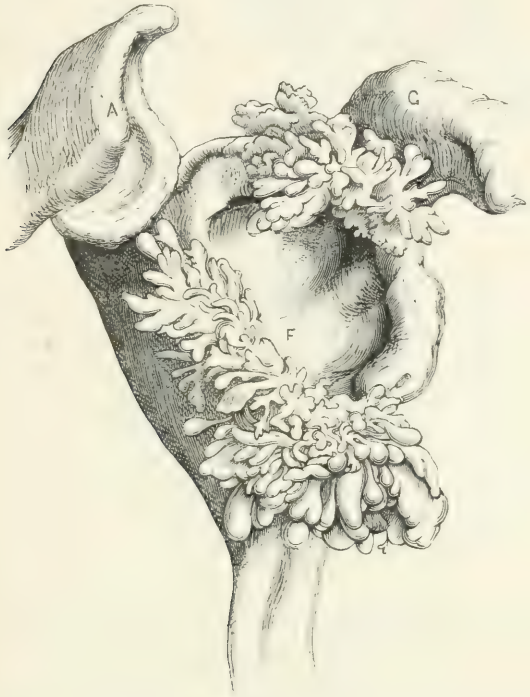
By J. Bland Sutton.

Johannes Müller in his work, 'Ueber den feinern Bau und die Formen der Krankhaften Geschwülste,' Berlin, 1838, describes briefly, under the name of "lipoma arborescens," pendulous fatty processes which hang about the synovial membranes of chronically diseased joints. These fatty procesees are nothing more than the villous fringes so frequently seen in joints affected with osteo-arthritis (rheumatoid arthritis) which have undergone fatty degeneration.

Typical specimens of this condition are very rare in museums, and I know of no drawing illustrating it in current literature. The

present specimen occurred in connection with the shoulder-joint of an old woman whose body was dissected under my supervision. Every joint betrayed evidence of osteo-arthritis. The shoulder-joints

WOODCUT 15.



The glenoid fossa of the scapula, showing "lipoma arborescens" (Müller).

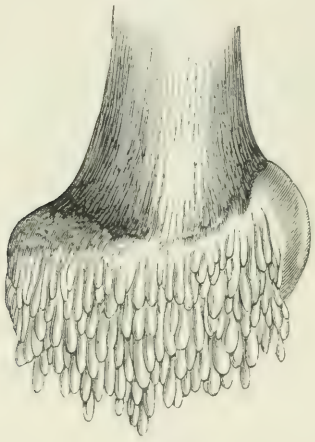
were most affected. Sprouting from the synovial membrane, especially near its attachment to the margins of the glenoid fossa, were numbers of pedunculated processes, many of them composed of fibrous tissue, but the bulk of them was made up of fat. The smallest of these villous outgrowths was of the size of a millet seed, the largest equalled in size a cherry stone. It was possible to trace in this specimen every gradation from the simple villous fringe to a pedunculated mass of fat.

The Museum of the Royal College of Surgeons possesses a very

typical example of this condition in connection with the knee-joint. It is described very graphically in the Catalogue thus :

"A section of the condyle of the femur, around the articular margin of which there is a crowd of small oval and branched growths of fibrous and fatty structures, each covered by a shining membrane, like a reflection of synovial membrane, and all attached by long slender pedicles. Their shapes and sizes are various ; some are like branching threads ; others are nearly cylindrical ; others flattened and lanceolate, &c.

Woodcut 16.



A femoral condyle beset with a crowd of fatty fringes. Lipoma arborescens of Müller. (Museum Royal College of Surgeons.)

Strictly speaking, the term lipoma ought not to be applied to this condition, for these pedunculated growths have nothing in common with the tumours to which the term is usually applied, except that they are made up of fat. Nevertheless the term lipoma arborescens is so apt and expressive that it is worth retaining.

January 3rd, 1888.

VII. MORBID GROWTHS AND TUMOURS.

1. *A case of diffuse symmetrical lipomata.*

By Walter K. Sibley, M.B.

The patient shown to the Society is a man (T. U—) aged 32, who during the past four or five years has noticed lumps appearing in various parts of his body. He states that the swellings on the right side of the occiput and beneath the chin began five years ago, about three months later that on the left side of the occiput, and those over the symphysis pubis appeared about the same time. For three or four years the abdomen has been somewhat prominent, especially in the upper regions. For about a year and a half the tumour in the leg and flank and the swellings about the arms have been present.

The patient, who is a potman, is a plethoric, well-nourished, muscular man with light brown hair and slight gluteal capillary injection about the face. Tongue slightly furred and tremulous. Thorax emphysematous, liver large, urine normal.

There are two swellings in the scalp symmetrically situated, one behind each sterno-mastoid muscle about the size of an orange, extending above as far as the superior curved line of the occipital bone, mesially to the ligamentum nuchæ and fading away below indefinitely. On either side of the spines of the upper three or four dorsal vertebræ there are other swellings.

There is also considerable fulness beginning above each zygomatic process and extending through the soft tissues of the neck and slightly into the face, meeting beneath the chin. Over the crest of the pubes is an ill-defined, slightly lobulated mass about the size of two oranges symmetrically placed, also on the left side between the lower ribs and the pelvic brim is a well-defined, some-

what larger lobulated tumour. There is also diffuse deposit of fat in the abdominal wall, the upper mass extending to the costal arch and below to some two inches above the umbilicus, and a second collection commencing just above the umbilicus and extending to nearly midway between this and the pubes. A small, very ill-defined lump is present over the lower ribs posteriorly on the right side. On each arm over the regions of the deltoid and of the heads of the triceps muscles is some general superficial fulness, also small masses just below the bend of the elbows on the flexor surface of the forearms. Also some indefinite swelling over the great trochanter of left side.

Most of the swellings have the same general characters. They are soft, those behind the scalp almost fluctuating, and with very indefinite margins; they are freely movable over the subjacent structures, the skin over them more or less adherent but otherwise unaltered; there is no pain or tenderness about them. The tumour in the left flank differs from the rest in that it is distinctly lobulated and well defined, is denser, and the skin over it is not adherent, and it is constant in its general appearance.

The skin in the lower part of the abdomen presents on either side of the rectus muscle some well-marked lineæ albicantes, these being at the edges of the lower abdominal mass. The same appearance of skin is seen over the region of the deltoid muscles, most marked on the left arm, the lines being more or less vertical.

The man states he is accustomed to drink considerable quantities of beer and spirits (Irish whisky) and that when in regular hard work, although getting more alcohol, the swellings become smaller.

The points of interest about this case appear to be that (as in those reported in the 'Medico-Chirurgical Transactions' for 1886 by Mr. Morrant Baker and Mr. Bowlby, and also in a case exhibited before the Medical Society by Dr. Kingston Fowler in the same year), the subject is a male about thirty years old, of decided alcoholic tendencies, and from their position and extent the swellings are superficial to the deep fascie, in fact, deposit of adipose tissue among the subcutaneous structures. It appears that most of the swellings alter considerably from time to time in size; notably is this the case with those on the occiput and that on the sides of neck and chin. So much does this one subside that he is able to shave with ease, which at other times he is unable to

do. They also become harder and softer at intervals. The asymmetrical one on the left flank, he states, never becomes smaller.

I have to thank Dr. Cecil Biss for allowing me the opportunity of exhibiting this case. *January 3rd*, 1888.

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2. *Large congenital lipoma situated between the abdominal muscles and fasciæ.*

By Frederic S. Eve.

History.—The patient, a well-nourished girl aged 13, was admitted to the London Hospital in July, 1887, with an immense tumour of the abdominal parietes. A swelling in the left inguinal region was noticed when she was four months old, and had since gradually increased in size. When two years of age she was seen at Guy's Hospital by Mr. Davies-Colley, who recommended removal, but the parents declined the operation.

Present condition.—There was a very large painless tumour, freely projecting from the front surface and left side of the abdomen. It extended from the costo-chondral margin to the groin, and from the middle line to the left flank. The skin over the tumour was freely movable, stretched, and of normal colour. The tumour itself was to some extent movable on the deeper structures, but was sufficiently attached to lead to the suspicion that there might be a prolongation from its deeper surface extending between the abdominal parietes. It was of softish, elastic, and uniform consistence, and at its inner margin large lobulations were felt. These, with its other characters and the history, determined the diagnosis of lipoma.

The circumference of the child's abdomen just above the iliac crests was twenty-nine inches. The diameter of the tumour from above downwards was twelve inches, from side to side nine inches.

Operation.—On dividing the cutaneous structures by an incision over its long axis, the surface of the tumour was found, to my surprise, to be covered by the extremely thinned aponeurosis of the external oblique. This was divided to a limited extent, and the surface and sides of the growth were then readily separated

from their surroundings. Towards the lower and inner part of its base was a lobe which dipped more deeply into the abdominal parietes and was adherent by tough bands of fibrous tissue to the fascia transversalis. The deeper part of the tumour contained a good deal of fibrous tissue, and it was probably by the growth of this and its subsequent conversion into fat that the increase of the tumour took place. After its complete removal the fascia transversalis was exposed to the extent of an area the size of the palm. Around this space were shreds of muscle-fibres which had been separated from the tumour. The internal oblique and transversalis muscles had either been penetrated or pushed aside in the growth of the tumour, which lay between the abdominal aponeurosis and the fascia transversalis. No relation could be traced between the tumour and the inguinal canal and round ligament, nor with the peritoneum. It probably originated in relation with the fascia transversalis, with which it was closely connected.

The remains of the abdominal aponeurosis and muscles were so thinned and separated that no attempt was made to bring them together.

The skin was united by sutures and a drainage-tube was inserted. Union took place by primary intention with the exception of a sinus, which discharged for some little time.

On the patient leaving the hospital there was no noticable protrusion of the abdominal wall at the site of the tumour, although the parietes must have been considerably weakened.

The tumour weighed $3\frac{1}{2}$ lbs., was slightly lobulated, but presented no peculiarity.

Remarks.—The chief points of interest regarding the case are the unusual situation of the tumour (namely, in the substance of the abdominal parietes, and springing probably from the fascia transversalis), its large size in proportion to that of the child, and congenital origin.

The interest as regards diagnosis turned on its relation to the abdominal parietes, and whether any part of it was intra-peritoneal or intimately connected with that membrane. In a list of cases of lipomata in unusual situations appended to a valuable paper by Mr. J. B. Sutton,[1] one case of lipoma is mentioned as lying between the pectoralis major and minor muscles ; another, recorded by Mr. Butlin, in which the tumour lay between the deep muscles

[1] 'Med.-Chir. Trans.,' vol. lxviii, 1885.

of the calf. It is needless to add that subcutaneous lipomata are common about the pelvic girdle and sacral region.

January 3rd, 1888.

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3. *A congenital cartilaginous tumour of the neck.*

By Frederick Treves.

A little girl, aged 3, was brought to me at the London Hospital in May, 1887, with a peculiar growth in the left side of the neck. The child was well developed and in perfect health. At her birth the confinement had been normal. The small mass in the neck had been noticed immediately after birth, and it had grown as the child had grown. The neck, the lower jaw, the hyoid bone, the larynx, and the pinnæ presented no deviation from the normal condition. There was no history of deformity in any member of the child's family.

On the left side of the neck a peculiar projection of the skin was visible. It looked and felt as if a foreign body were lodged beneath the integuments. The little elevation was ridge-like and was raised about one centimetre above the general level of the surface. On examination a hard elongated body was made out. It was of irregular shape and about an inch in length. It felt like cartilage and could be bent under the fingers. The skin covering it was perfectly normal. There was no tag-like arrangement of the integument, no growth in the skin, and no sinus in the vicinity. The foreign body was very mobile but could not be entirely separated from the skin.

The mass was placed obliquely in the lower part of the neck. It occupied the lower third of the anterior triangle; was parallel to the great cornu of the hyoid bone, and was midway between the upper margin of the thyroid cartilage and the clavicle. It was over the sternal part of the sterno-mastoid muscle. The upper extremity (A) pointed to the left ear, the lowest end (B) to the sterno-clavicular point. A ridge (C) between these points caused the elevation of the skin.

I excised the mass. It was quite subcutaneous and had but a feeble attachment to the skin. It was very movable. It proved to be a mass of cartilage embedded in connective tissue. From the most deeply placed part of the cartilage (D) a fibrous cord passed, taking a direction apparently towards the spine. No vessel required ligature. The muscle was not exposed, and the wound healed by first intention.

The mass on removal was found to be made of cartilage. It took the form of a rod that was twisted upon itself at its upper part and at its lower extremity was divided into two plates (see Woodcut 17).

Woodcut 17.

I am not aware that many cases precisely similar to this have been placed on record.

There is little doubt but that the nodule was developed about the fourth branchial cleft.

The congenital fistulæ that are so frequently left at the neck from imperfect closure of the branchial clefts would appear to most usually concern the first and the fourth clefts.

The sinus in the latter situation has exactly the position and direction assumed by this piece of cartilage.

The fistula is found to open over the lower third of the sterno-mastoid muscle from 1 to 2 cm. above the sterno-clavicular joint. It lies near the sternal part of the muscle and may pass in the direction of the spine to a considerable depth.

Heusinger ('Virch. Arch.,' xxix, p. 365 ; xxxiii, p. 175) and others have discovered fragments of bone and of cartilage in the depths of such fistulæ.

In the great majority of instances, however, in which cartilage has been found that substance has been embedded in loose tags

or pendulous processes of skin that occupy the site of the usual fistula but that are associated with no actual sinus. Virchow ('Ueber die Entstehung des Enchondroms,' Berlin, 1875) has described these strange masses, and a very full account of them has been more recently given by Weinlechner (Gerhardt's 'Handbuch der Kinderkrankheiten,' Band 6, p. 114, 1887) under the title of "Kiemenganghautauswüchse mit knorpeligem Gerüste." These cartilage-containing skin tags have been observed five times on the left side of the neck and three on the right. They do not appear to be met with on both sides at one time, as is sometimes the case with the branchial fistulæ. It is noteworthy, also, that the latter sinuses are more common on the right side of the neck than the left. The cartilages contained in the integumentary tumours are never rounded but are—as in the present case—flat and angular.

The interest of the present instance depends upon the circumstance that there were no changes of any kind in the skin, and but for the projection of the surface the presence of the cartilage nodule would not have been suspected.

A peculiar case, a little bearing on the present subject, is reported by Mr. Muirhead ('British Medical Journal,' vol. ii, 1887, p. 177). It concerned a child five weeks of age. "Extending from below the left ear to the left upper corner of the sternum is a firm, broad strip of cartilage, probably already ossified in part, resembling in shape and size the sterno-mastoid muscle, but presenting a forward projection in the middle of the neck as if bent at an obtuse angle. It may be caught between the finger and thumb and moved freely from right to left. The cartilage is perfectly separate from the jaw above and from the clavicle below it, and seems to have no other connections except those described. Its origin from the temporal bone is immediately below and within the left auricle, about half an inch in front of the normal origin of the sterno-mastoid muscle. The probability is thus suggested that its upper attachment is connected with the styloid process. Its connection with the sternum occupies the position of the sternal portion of the sterno-mastoid muscle."

It is hardly possible that this peculiar cartilage could have been connected with the branchial clefts. It seems rather to have been associated with the sterno-mastoid muscle.

November 1*st*, 1887.

4. *Enchondroma of hand.* (*Card specimen.*)

By H. A. Lediard, M.D.

From a young man, aged 22, who had had enlargements of the fingers as long as he could remember, but increasing in size of late.

On the index and middle fingers of the right hand are rounded, firm tumours, the size of large and small walnuts, which project from the sides of the first and second phalanges. On the dorsum of the hand and at the base of the index and middle fingers is a tumour the size of an egg. There are also two tumours placed laterally on the first phalanx of the thumb, but easier felt than seen. More particularly, the first phalanx of the index finger has two tumours, whereas the second phalanx shows but one. The first and second phalanges of the middle finger show two tumours each. The growths on the second are smaller than those on the first phalanges. The hand could not be closed and was practically useless, and the index and middle fingers were amputated, together with portions of the metacarpal bones adjoining.

The left hand showed similar growths in the middle finger and thumb, but in too early a stage to cause any trouble.

On splitting the fingers down it was seen that the terminal phalanges were healthy, and that the new growth had originated in the medulla of the affected bones. In no case was the normal cartilage of the joints altered. More closely seen, there was but a small amount of natural osseous tissue left, as the shafts and proximal ends of the phalanges were expanded by the presence of cartilaginous tissue having a pink and grey gelatinous appearance. The metacarpal bone of the index finger was more diseased than that of the middle finger and from it grew the tumour on the dorsum of the hand already described.

Microscopic examination showed chiefly hyaline cartilage, the nuclei of which were branched or stellate whilst the cartilage cells were both scattered and crowded. *February* 21*st*, 1888.

Fig. I

Fig. III

Fig. II

DESCRIPTION OF PLATE XIX.

Fig. 1 illustrates Mr. Targett's specimen of Bony Tumour of the Buttock. (Page 304.)

Fig. 2 illustrates Mr. F. Treves's specimen of Sarcomatous Tumour of the Sole of the Foot. (Page 308.)

Fig. 3 illustrates Dr. Wilks' case of Furrows on the Nails. (Page 370.)

From a photograph.

5. *A case of mixed tumour of the parotid.*

By Thomas F. Chavasse, M.D.

Sarah N—, aged 45, admitted to the General Hospital, Birmingham, in May, 1886.

There is nothing calling for particular note, either in the family or the personal history of the patient until the age of fifteen. A lump the size of a pea was then first noticed immediately below the right ear; this gradually increased to the size of a marble, and then remained stationary for fifteen years. At the expiration of that time, the swelling again commenced to slowly increase in size, and in 1880 it had reached the size of an ordinary duck's egg. An attempt was then made by a surgeon to remove the growth, but according to the patient's statement (which is confirmed by her medical attendant, Mr. Hart, of Harborne) the operation was not completed, owing to the severe hæmorrhage which was occasioned. The wound was closed and healing took place. After this the tumour began to grow rapidly, and caused pain by its weight in the neck and shoulder on the side affected.

On admission to the hospital there was a tumour as large as a fœtal head, filling the right parotid region and extending downwards in the anterior cervical triangle. Over its surface a good many large veins were seen coursing, and a cicatrix three inches long, the result of the previous operation, existed. With regard to the deeper structures of the neck, the growth appeared freely movable. Generally speaking, it was firm to the touch, but at the upper part it was distinctly nodular, and at the lower it was semi-fluctuating.

Operation.—On May 18th, removal was effected by a longitudinal incision extending from the top to the bottom of the growth. A distinct capsule was present, and no deep connections calling for special notice existed. The bleeding was fairly copious but readily controlled by catch forceps. Corrosive sublimate dressings were applied, and on June 19th the patient went home with the wound quite healed. She has remained well up to the present time.

On removal, the tumour was found to weigh 1 lb. 4 oz.; it was ovoid in shape and furnished with a distinct capsule.

On section, it was composed of a reddish grey, fleshy-looking tissue of firm consistence. Embedded in its substance, especially beneath the capsule and in the cortical parts, were small nodules of hyaline cartilage, surrounded more or less by a zone of soft gelatinous tissue. In no part were any distinct cysts.

Histologically.—The excellent sections exhibited were made by Dr. Crooke, Pathologist to the Hospital. The drawings are by Mr. Teichelmann. The growth is a mixed one. It consists mainly of lobuli presenting the structure of fibro-adenomata. The amount and character of the stroma vary; in some parts it is mucoid in type, contains spindle-shaped nuclei disposed between anastomosing columns of cells of the growth, which sometimes form quite a network.

In other parts the stroma is more fibrillated, contains blood-vessels, and surrounds loculi or acini lined by the cells of the growth, and, in such parts, the acini are better defined and the cells are placed perpendicularly to what looks like a basement membrane.

In other parts the stroma is denser, fibrous, and branching, sending out processes across the acini between the cells, and the growth here resembles a fibro-adeno-sarcoma.

Regarding the cartilaginous portions, in some parts the matrix is quite hyaline in character, the cells being surrounded with a well-defined capsule that stains deeply with eosine and hæmatoxylin. Some of the capsules are very large, others are withering, and the contained cells degenerating. In other places the matrix is becoming fibrillated and gradually degenerating into myxomatous tissue, and on the transition borders many of the capsules of the cartilage contain branched cells.

The myxomatous portions are well defined, and at their margins are seen adherent cells in the form of branching or clavate processes, with individual, branching, myxomatous cells between, so as to resemble a myxosarcoma, until presently cell masses are met with resembling those within the acini of the adenomatous parts. The myxomatous change, therefore, in the cartilage and in the stroma of the growth seems to be a secondary and a degenerative process.

The atrophy of the lobular acini of the adenomatous part by the development and growth of the cartilage in the stroma is well seen at the peripheral part of the tumour.

The record of this mixed tumour of the parotid brings prominently forward the question of the origin of the cartilaginous cells. It has been suggested that, owing to traumatic or other causes, subacute parotiditis may give rise to cells which become transformed into cartilaginous elements. Another, and to my mind a more plausible theory, is that in early fœtal life some of the cartilage intended for the ear, from some cause or other, gets stranded in the parotid, and after a latent period makes its presence apparent. As might be expected, the effects become evident during the growing period of life. The few patients, all of whom have been women, that have come under my immediate notice with so-called enchondroma of the parotid, have noted the first signs of tumour formation before or about the period of puberty.

If the hypothesis of this stranding of fœtal cartilage be correct, the presence of this abnormal constituent may act as an irritant and cause an hypertrophy of the true glandular elements of the parotid. An examination of the microscopic sections shows that in this case, when plenty of time for development had been allowed before the removal of the growth, perfect adenomatous tissue is abundantly seen.

The presence of myxomatous tissue in the tumour is probably the result of retrograde changes, in which the higher type of connective-tissue growth degenerates into a lower form. The same may be said to hold good with regard to the yet more embryonic elements, viz. the sarcomatous.

An examination of the various sections of this tumour tends to confirm the belief that the theory is both probable and possible.

As to recurrence after removal, no hard-and-fast rule can be laid down, but any return of such a growth appears to be slow. In the present instance nearly two years have elapsed, and there are no signs of any new formation.

In a case recorded by Mr. Sidney Jones in vol. xxiii of the 'Transactions' of this Society, a growth somewhat similar on section, but much larger, weighing 3 lbs. 4 oz., was removed from a man aged 58. Twenty-eight years before a tumour the size of a walnut had been excised from his parotid, and then there were no symptoms of recurrence for nearly five years.

Nine years ago I removed from the left parotid region of a girl, aged 18, a mixed growth, the size of a bantam's egg, with cartilaginous nodules well marked, but with none of the sarcomatous

intercellular substance evident, as in the case of Sarah N—, and up to this date there has been no return of the tumour.

In these instances of tumours of the parotid gland containing cartilage may not the microscopic appearances of the other parts of the neoplasm furnish some sort of clue as to the future clinical history? If, as in the case we are now discussing, the sarcomatous elements are abundant, then the ultimate prognosis is probably unfavorable. But if the suggestion of retrograde metamorphosis be accepted as producing the sarcomatous tissue, then the earlier such a tumour be removed the better for the patient, the more favorable the prognosis.

The age of the patient and the family history must also be taken into account. Sir James Paget is of opinion that in cases of cartilaginous tumours in which the cartilage is of a soft type, recurrence is very apt to take place, especially in those with a cancerous family history. *February* 21*st*, 1888.

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6. *Bony tumour of buttock* (*? ossification in bursa.*) (*Card specimen.*)

By J. H. Targett, M.S.

[With Plate XIX, fig. 1.]

History.—George E—, aged 23, iron ship-builder, works in a sitting position with a sliding motion which he compares with that of rowing. Has followed this occupation for twelve years at ten hours per diem.

Four years ago he noticed a small lump over the left tuber ischii which grew very slowly at first, but more rapidly during the last two years. It caused some discomfort after sitting on it for any length of time. The tumour was removed, and found to be embedded in the abundant fat of the buttock, but adherent by its deeper surface to the lower edge of the gluteus maximus. It was hemispherical in shape, and measured four inches in diameter and three inches in depth.

Description of tumour.—A vertical section shows a number of

masses of bone embedded in fibro-fatty tissue; their cut surfaces are somewhat circular and have a distinct narrow border of cartilage, the chief portion of the masses being composed of dense osseous tissue. The majority of these bony masses appear to be quite independent of each other, for they admit of slight lateral movement; moreover, some of them have a well-defined synovial sac reflected over a large part of their circumference, in which they play like a joint. The smaller nodules are entirely cartilaginous, and the deeper border of the specimen is composed of very dense fibrous tissue to which muscular fibre is adherent. The convex surface of the specimen is a little bossy from the protrusion of these masses of bone. There is no sign of capsule to the tumour, nor of any cavity in its interior.

Microscopically the smaller nodules are composed of hyaline cartilage, and the larger ones of dense true bone, bounded by a zone of ossifying cartilage as described above.

Remarks.—This tumour can hardly have arisen in relation with the normal ischial bursa, as it was quite superficial to the gluteus maximus; but the history of long-continued irritation makes it probable that it started in a similar, though adventitious, structure.

April 17*th*, 1888.

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7. *Sarcoma beneath the pectoralis major.* (*Card specimen.*)

By Stephen Paget.

This growth was removed from a gentleman aged 69. It was first noticed four months ago. It grew with very great rapidity to the size of a cocoa-nut: an elastic solid mass, movable on the ribs, lying beneath and behind the right pectoral muscle. The skin was not adherent over it, nor were any enlarged glands to be felt. The mass was exposed by a free incision through the muscle, and removed. It was encapsuled, lying on the ribs, but not closely adherent to them. It was soft, cystic, gelatinous, hæmorrhagic, and almost broken down in some parts. The wound healed quickly.

The disease recurred round the scar with very great rapidity, and was twice again removed, but it grew very speedily, and the patient died from exhaustion in June, 1888.

The structure of the first growth was that of a mixed-celled or fibro-sarcoma. The recurrent growth was softer, almost diffluent.

December 20th, 1887.

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8. *Multiple sarcomata of skull, ribs, and glands.* (*Card specimen.*)

By J. H. Targett, M.S.

History.—Edgar G—, aged 1 year, fell out of a chair two months ago and grazed his nose. Soon after a swelling developed in left frontal region which steadily increased.

On admission, hemispherical swelling three inches in diameter, elastic, non-pulsatile. Veins dilated over it. No brain symptoms. No history or signs of syphilis.

On tenth day two other small tumours noticed on the head. Temperature raised to 101° for last three days. The child is now rather drowsy, and has rapidly wasted.

Death on twentieth day after admission from broncho-pneumonia. The tumours have grown rapidly of late.

Autopsy.—There is a large mass occupying the middle of left frontal bone, spherical in shape, two and a quarter inches in diameter, projecting to like extent on both external and internal surfaces of cranium. Pericranium closely adherent and forms a capsule on the one side, while the dura mater is tightly stretched over inner surface but not definitely adherent. On section soft, dark red in colour; a knife will pass readily through centre of growth, the bone being extensively invaded. There are three or four small secondary deposits in the cranium, in the neighbourhood of the main tumour; one of these is quite in the centre of the bone, and only visible on holding skull up to the light. Another deposit was found in left lesser wing of sphenoid, and a much larger one in left temporal region.

There was a considerable depression in first and second left frontal convolutions due to the main tumour.

Viscera healthy, except a small nodule in liver.

Several ribs on both sides invaded at their sternal ends, the bone

being almost entirely replaced by growth. This is of interest, as there were rickety changes in other parts. There was a large mass of growth along the dorsal spine due to deposits in the intercostal glands, but it had not involved the spine. Below, this was continuous with a similar mass in the abdomen which reached the whole length of lumbar spine, and on the left side to spleen, tail of pancreas and kidney. The latter was almost encapsuled with growth, but its structure was healthy, and cortex not adherent to growth. The left ureter passed through the mass of growth, and must have been compressed, for the pelvis was dilated above. On section, this large abdominal mass was found to be deposits in the mesenteric and lumbar glands, which were much enlarged and closely applied to one another. The mesenteric and other vessels were embedded in the growth.

Microscopically the frontal tumour consisted largely of masses of cells embedded in blood-clot, from extensive extravasation of blood. The firmer portions had a somewhat alveolated arrangement. It was a round-celled sarcoma, the cells being very uniform in size.

January 17th, 1888.

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9. *Melanotic sarcoma, growing from the edge of the nail of the index finger.* (*Card specimen.*)

By H. A. Lediard, M.D.

This small melanotic tumour was found growing at the side of the nail of the index finger of a woman aged 40. The surface was firm, and resembled granulation tissue, no trace of pigment being visible. The tumour had been growing, almost painlessly, for two years. The nail was torn off, the growth snipped off with scissors, and the phalanx thoroughly scraped. Microscopic examination proving that the growth was a melanotic sarcoma, the finger was amputated at the metacarpo-phalangeal joint a few days later.

The patient came first on May 27th, 1885, and died during July, 1887, with a large number of melanotic tumours over the body.

The original growth shown measures three quarters of an inch

long, half an inch broad, and three quarters of an inch deep. To the naked eye a large quantity of pigmented growth is seen more especially on the deep surface of the tumour.

October 18*th*, 1887.

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10. *Sarcomatous tumour of the sole of the foot.*

By Frederick Treves.

[With Plate XIX, fig. 2.]

The patient from whom this specimen was obtained was a man, aged 56, a labourer, who was admitted into the London Hospital in May, 1886, with a large tumour growing from the sole of the left foot. The growth formed a remarkable projection. It was rounded and particularly well circumscribed. It was a little flattened on its plantar aspect and slightly constricted at its base. It was even and of regular outline and to the touch firm and resisting. The skin covering the growth was greatly thickened but in other respects normal. The mass was limited to the sole, and its base corresponded to the posterior part of the fifth metatarsal bone. It had a circumference of ten inches, measured about three inches in the transverse direction and three and a half in the antero-posterior. It projected one and three quarter inches from the surface of the sole. The part of the tumour nearest to the great toe was a little more prominent than the rest and decidedly softer. The tumour had been first noticed thirty years ago and made its appearance after a severe attack of rheumatism. Its growth had been steady but exceedingly slow. The man had always walked upon it, placing the foot flat upon the ground. He walked indeed mainly upon the heel and the tumour. For many years the presence of the mass had caused him to walk lame, and he had been in the habit of cutting away the outer part of his boot to allow the tumour to project.

He had enjoyed fair health all his life and the foot had given him no particular trouble.

During the last two months the mass had commenced to grow more rapidly, the growth showing itself in the softer and more prominent part of the tumour to which allusion has been made. I removed the tumour and ascertained that it grew from the plantar fascia below the fifth metatarsal bone. The wound healed rapidly.

On section, the greater part of the mass was perfectly homogeneous, was pale and dense, and exhibited the ordinary appearance of a fibrous or fibro-cellular tumour with a very well-defined capsule. The part of more recent growth appeared as a rounded mass the size of a horse chestnut that was pretty clearly defined from the rest of the tumour. It was less perfectly encapsuled, was much softer, was of a yellowish pink tint and much more succulent. It had in short all the appearances of a sarcoma.

Briefly it may be said that the older part of the growth showed under the microscope the structure of a fibrous tumour, the more recent part that of a spindle-celled sarcoma.

Clinically, the case suggests that age had apparently in this instance more to do with the development of the sarcomatous change than had other factors. The tumour had for years been exposed to irritation, to great fluctuations of temperature, and to no moderate degree of violence, for with every step the patient took the mass must have been crushed beneath the weight of his body. Yet for thirty years these supposed excitants of tumour growth had been acting without hindrance, but it was not until the man had become old that any change took place in the character of the growth.

A greater interest, however, attaches to the precise nature of the change that occurs when an innocent tumour "takes on malignant action." Such change is common enough. The long irritated wart may in time develop into an epithelioma. The fibrous or fibro-cellular tumour may, in what would appear to be a like manner, assume the characters of a sarcoma. The simple adenomatous tumour of the breast may in the course of years take upon itself the features of a cancerous growth. It is probable that a like metamorphosis may be concerned in all these instances. In detailing such change it is commonly assumed that the primary innocent growth becomes in some part of its substance actually converted into the tissue of a malignant neoplasm.

It is not unusual to say that the tumour has undergone malignant degeneration, or that it has taken on malignant action. Whatever

expression, however, is employed, the assumption follows that it is the actual tissue of the innocent tumour that has undergone change. If such change be possible the title neoplasm would seem to be a misnomer. The growth is not so much a new growth as the product of a transformation in already existing tissue.

I am under the impression that in cases of so-called malignant degeneration the primary tumour remains—so far as its structural elements are concerned—unchanged, and that the condition is simply that of a tumour of a tumour. The primary growth will suffer from the vicinity of the malignant mass just as normal tissues will suffer, and the growth will be probably involved in the destruction that follows in the wake of the ill-disposed neoplasm. The examination of the present specimen certainly appears to support this view.

To the naked eye the sarcoma is a clearly defined new deposit in the substance of the ancient tumour.

The patient was aware of this new growth, its locality was clearly indicated before the operation was performed, it differed from the primary growth in aspect and consistence, and its relation to the fibroma would appear to the naked eye to be well expressed by the term a tumour of a tumour.

The microscopic examination provides still more substantial evidence. The main tumour is simply a fibrous growth, singularly uniform in appearance and structure in all parts.

A section of the central portion of the newer growth reveals the typical formation of a spindle-celled sarcoma of the most ordinary type. When the boundary line of the sarcoma is reached, it can only be said that the two tumours meet.

The sarcoma is not encapsuled. The fibroma forms its capsule wall. The periphery of the malignant tumour shows a less orderly structure than does the centre. The cells are less methodically arranged, they are of less regular size and of more uncertain shape. The growth would indeed appear to be disorderly. At many points the sarcoma and the fibroma have blended,—the one has grown into the other; but at no single point is there the least evidence to show that the sarcoma has been developed from the fibroma tissue. The two growths remain everywhere distinct. There is nothing to suggest that a single sarcoma-cell has taken its origin from an existing fibroma-cell.

It would appear that the primary growth has been invaded by a

sarcoma, and not that a portion of its substance has undergone any process of conversion.

The specimen may be taken therefore as an example of a tumour of a tumour. *December 20th*, 1887.

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11. *Case of villous sarcoma of the neck and heart.*

By Joseph Griffiths, M.D.

I am indebted to Mr. Carver for his kind permission in allowing me to bring this case in full before the Society, and to Mr. Francis for the clinical notes.

Ellis A—, aged 47, carpenter, admitted into Addenbrooke Hospital, October 23rd, 1886, under the care of Mr. Carver.

Three months prior to admission patient noticed a tumour, the size of a hazel-nut, movable, and situated about the middle of the posterior border of the left sterno-mastoid muscle. Suffered from headache the last six weeks. The tumour has rapidly enlarged of late.

Father alive and well. Mother died at age of sixty-five; cause of death not ascertained. Has brothers and sisters alive and well. There is no history of new growth in the family and no evidence of syphilis.

A pale emaciated looking man with sunken eyes, cheeks, and temples. Has always, previous to present illness, enjoyed good health, except a little rheumatism at times.

On left side of neck there is a large, hard, very prominent, nodulated swelling, apparently implicating sterno-mastoid muscle, or immediately subjacent and stretching it. It extends from corresponding mastoid process downwards and forwards, below ear, to the left angle of lower jaw, reaching to nearly sternal end of sterno-mastoid muscle. The lower half is less prominent and easily made out. The mass is fixed, and the skin is tightly stretched over it at one part where there is a certain amount of redness. The tumour is equal in consistency throughout, and feels like a mass of enlarged glands. The external jugular vein of that side

cannot be made out. In front of the neck, a little to the left, just above the manubrium sterni, is another isolated growth of the size of a walnut. No pain or tenderness. Neck is stiff, and its movements are restricted.

There is difficulty in swallowing solids only, but no pain during act. Nothing detected by buccal examination and no blood from pharynx.

He also suffered from a slight cough, and brought up some very offensive sputum. No pain in thorax. Urine free of albumen and of sugar.

Further notes were not taken, and the patient died on January 26th, 1887.

Post-mortem examination.—A large, irregular, and firm mass involves, and extends between, the muscular fibres of the upper third of the left sterno-mastoid muscle, gradually lost above in cord-like processes reaching up to the skull in and between the sterno-mastoid and the adjacent muscles. In front of the mass, occupying apparently the lumen of the vein, is a thick, cylindriform structure, half an inch in diameter, which at the level of the hyoid bone rapidly enlarges to about an inch in diameter, and passes downwards for two inches, then it rapidly narrows into a thin filament. At its upper enlarged end it is connected on its inner aspect by a constricted portion to another cylindriform structure of the same size, lying in the position of the external jugular vein, and reaching to the left subclavian; this portion was the part felt as an isolated mass of supposed lymphatic glands in the living subject. At the junction referred to there is on the inner side of it a small irregular mass, and from its posterior part can be traced downwards along the left border of the thyroid gland a cord-like structure, the size of an ordinary quill, which gradually becomes thinner, and terminates near the lower border of the isthmus; and upwards from it three or four similar cords can be traced between the extrinsic muscles of the tongue, where they become gradually lost. The left internal jugular vein is distended in the same manner as the external jugular from the level of the hyoid bone downwards. Above this point the lumen is free and the vessel walls are healthy.

The mass in the sterno-mastoid on section was seen to consist of round or ovoid masses of a whitish tissue, varying from a very small size to about half an inch in diameter, into some of which

small hæmorrhages have taken place. Each was surrounded by a distinct, well-defined, fibrous capsule, which appeared to be the wall of a small vein. The growths in the external and internal jugular veins are identical; each consists of very numerous, delicate, sometimes flattened, and irregularly branching filaments, varying in width from 1 to 2 mm., which are very closely packed, so as to form a dense solid mass, filling more or less completely the lumen of the vessel. Occasionally small masses of fibrin are seen between the filaments. The mass in the lumen is connected to the interior of the vessel wall by means of delicate, flat, band-like processes, which are not very numerous, and they were not easily separated from the vessel wall. The filamentous nature of the growth is well seen at the divided ends of the internal and external jugular veins. Unfortunately, being ignorant of the nature of the case, I had removed the heart and lungs in the ordinary way, and thereby disturbed any continuity of growth that may have existed from the root of the neck towards the heart. There was nothing found in the vena cava superior nor in the right auricle, but in the cavity of the right ventricle was a cluster of "stunted, shrub-like, leafless growths," I judge like that so described by Sir James Paget in a somewhat similar case. They were entangled in fibrin, and also between the chordæ tendineæ. Here there existed no filamentous connection with the wall of the ventricle, musculi papillares, or chordæ tendineæ; so far as I could determine, therefore, it was loose in the cavity. The branches of the shrub-like growth are of different sizes, tubular and dilated here and there into cysts containing clear limpid fluid. On the exterior of the tubes were small projections or branches which in many cases form cysts. Extending along the pulmonary artery is a slender irregular cystic growth, 1 to 2 mm. in thickness, and in each of the divisions of the pulmonary artery a similar growth is seen. There was no continuity between these growths and that in the ventricle. In the lumen of one of the branches of the left pulmonary artery another of these cystic growths, partially filling it, can be traced from the root to the periphery of the lung, where some of the lateral terminal branches are also filled with a continuation of the growth. Delicate filamentous attachments between the growth and the vessel exist here as in the veins just mentioned. There is no evidence of the formation of a thrombus in any part of the vessel, and the external appearances and minute structure of the growth are quite different

from those of a thrombus. No other changes in this lung. In the first divisions of the right pulmonary artery are similar growths which almost occlude some of them; and in this lung there is a large gangrenous cavity towards the middle of its substance.

The nervous system not examined. All other organs and tissues healthy.

Microscopically, one of the ovoid or round masses in the sterno-mastoid, under a low power, is seen to consist of a capsule or vessel wall, and filled with dendritic cell-growths, in places adherent to the wall, and in these intervening spaces blood was found. The vessel wall contains no muscle-cells. Under a higher power each of these dendritic processes exhibits at its periphery two or more layers of flattened spindle-shaped cells, and in the interior were irregular shaped cells, varying much in size, with a good deal of intercellular substance approaching that of connective tissue. In many of the processes I found near the centre one or more giant-cells, and as the question of the formation of "giant-cells" is of much interest to the pathologist it may not be out to place to consider their formation in this case, which could be pretty clearly traced to be as follows: A particular cell enlarges in its nucleus and protoplasm, both of which appear at this early stage otherwise unaltered. During further enlargement the nucleus becomes finely granular, more or less opaque, and its outline irregular and indefinite; the surrounding protoplasm undergoes a similar change. Later, adjacent cells similarly affected unite, and some of the nuclei appear to unite also, and to constitute a large, many nucleated, ill-defined mass. A section of one of the branches of the shrub-like growth from the right ventricle shows a cyst with a thick wall, which consists of cells with much intercellular substance and of a structureless material in small patches, varying in amount. On the exterior of the growth is a little coagulated fibrin in places. This structureless material led me to examine for the presence of micro-organisms, but after several attempts by various methods I failed to discover the presence of any.

The case I have thus ventured to bring before you being of an unusual nature led me to investigate the literature. I have, however, been able to find only two cases, and these present in a measure only similar conditions.

One is by Sir James Paget, described and figured in the 'Med.-

Chir. Trans.,' vol. xxxviii, which began as an enchondroma of the right testis, and proved fatal by producing secondary cartilaginous growths in the lungs, and also growths in the vena cava inferior and pulmonary arteries, similar, apparently, in nature to those in the case before you.

Sir James is of opinion that the growth extended from the testis towards the vena cava through lymphatic channels which increased in size and dilated to accommodate themselves to the growth. In my case the growths, small and large, were distinctly in vascular channels, which are venous in nature. Also, in my case there is considerable evidence in favour of the view that the growth originates from the interior of the veins, but that, I think, must remain a doubtful point for the present.

The other case is by Prof. Weber in 'Virchow's Arch.,' Bd. 35, S. 501, in which enchondroma of the pelvis existed at first, the skeleton being affected in many parts. Shrub-like growths in the veins of the pelvis and also in the pulmonary arteries were present, of which he gives a drawing.

These two cases originated as enchondroma, and in a later stage in their development assumed a dendritic form of growth in the vessels, whereas in my case no such origin exists, and the microscope reveals a dendritic mode of growth in what I conceive to be the seat of origin.

This tumour suggested many other points of considerable interest in general pathology into which it is not my intention to enter in the present paper. *April* 17*th*, 1888.

Report of the Morbid Growths Committee on Dr. Griffiths's specimen.—One of the most important facts in regard to this highly interesting specimen is that the whole of the morbid growth is contained within the vascular system. This relation is obvious with respect to the greater part of the tumour, and careful dissection shows that the large solid mass in the neck, extending between the muscular fibres of the upper third of the left sterno-mastoid muscle, is constituted by a plexus of veins which are enormously distended with similar new growth, and that what appear in the section as "lobes" of the tumour are in reality closely packed and distended veins; the walls of the vessels admit of being defined in this situation, and the "lobes" of the new growth are traceable into many of the veins, which spread out

from the plexus, and which are filled with similar morbid substance.

The occurrence of intravascular growths in veins, although rare, is well known. It is not infrequently to be observed in malignant disease of the kidney, when the growth may extend into and distend the renal vein. And there is a specimen in the museum of St. Thomas's Hospital (C. 218[4]) of distension of the cervical veins by an infective tumour, which recurred after excision of the scapula.

In all these cases, however, as well as in that recorded by Sir James Paget and that by Weber, which are noted by Dr. Griffiths, the growth has occurred by extension from another seat. In the present instance the whole of the growth is contained within vascular channels, and in this regard is, so far as we know, unique.

We need only add to the author's careful account of its histology that the tumour possesses a proper vascular supply. It is obvious that by means of this the nutrition of the growth has remained unaffected notwithstanding that all circulation through the lumen of the containing vein has long since ceased. There is no such central relation of the vessels in the processes of the growth as would justify the tumour being classed as an angiosarcoma.

The cystic and tubular condition of the growth within the heart and some of the branches of the pulmonary artery are due to colloid or mucoid change in its central portions. In the walls of such hollowed parts there occur areas of degeneration, the cells of the growth being especially the seats of the change; these become swollen and converted into a homogeneous refractive substance staining deeply with carmine. The formation of secondary cysts in the wall of the primary is explicable in the same way, by the softening of cell-groups in the tumour substance.

This tubular condition of long portions of the growth is so curious as to have suggested to us at first that the structure might be a parasite. It is clear, however, that these portions are histologically of essentially the same nature as the rest.

That the growth has originated from the vessel wall is demonstrated in certain of the sections by the fact that lines or groups of the cells are to be seen in the fibrous tissue which forms the inner parts of the thickened wall of the vein; the growth nowhere extends through the entire thickness of the latter. Whether the

tumour has originated in one spot or in many we do not think can be determined, nor can its precise mode of extension.

In some places the growth has for long distances no connection with the vessel wall; in others delicate adhesions exist, and in others again, as before noticed, the growth is inseparably incorporated with the wall of the containing vein. It is not improbable that by means of secondary adhesions arising between the advancing growth and the vessel wall the tumour may have received an accession in its blood supply.

The presence of so conspicuous a number of "giant-cells" is noteworthy. And it may be suggested that this is explicable here, as in the case of myeloid sarcoma of bone, by the inherited potentialities of the parts from which the tumour has grown. The giant-cells in the central tumours of bone may be associated with their normal presence in the medulla from which the tumour originates; and in a tumour arising purely in connection with vessels, the existence of such cells may be associated with what is known of vascular histogenesis, in which process multinucleated or vasifactive cells play so prominent a part, and the canalisation of the tumour possibly marks an alliance with what also occurs in the vasifactive process.

The author states that from the homogeneous appearance of certain parts of the growth he was induced to look for micro-organisms, but was unable to detect any. The presence of giant-cells and other characters of the growth might suggest the same inquiry. But the new growth has not the characters of any of the *ordinary* infective granulomata, though it may nevertheless be locally infective; nor is it impossible that it has spread, in part, by a process of auto-inoculation similar to that by which the peritoneum becomes infected in cases of papilliferous ovarian cysts which have ruptured into its cavity.

R. W. Parker.
Samuel G. Shattock.

12. *Nævoid growth removed from scrotum.* (*Card specimen.*)

By E. Bellamy.

The patient is a young man, aged 21, who had a congenital nævus of large size in the left side of the scrotum. He desired to enter the army, but it was diagnosed to be variocele, and he was refused.

I made a free incision through the integuments, reached the vascular growth, and carefully dissected it away; it had no apparent connection with the cord, and almost "shelled" out. But few vesicles required arrest. The specimen on section shows a mixed form of angioma, with some fatty degeneration between the vascular masses. The patient made a rapid recovery.

November 1*st*, 1887.

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13. *Large subcutaneous nævus from the parotid region.* (*Card specimen.*)

By H. A. Lediard, M.D.

This large nævus was removed from the parotid region of a child six months old.

About a month after birth a swelling was observed, which gradually increased, without change in the colour of the skin over it, so that the nature of the tumour was not at first suspected.

Examination subsequently proved that the growth was nævoid, but from the lobulation of it it seemed probable that it was a nævolipoma, hence the reason for excision.

The tumour was fairly well encapsuled, but bled freely when this covering was torn. Large vessels are seen to enter the deep surface.

Microscopic examination failed to show anything but ordinary nævoid tissue.

Considerable facial paralysis resulted, but the child made a good recovery.

The nævus measured three inches long, two inches broad, and one inch thick.

In the 'Transactions,' vol. xi, p. 267, Gascoyen records a case of nævus involving the parotid gland and causing death by suffocation. *October 18th*, 1887.

P.S.—Further examination showed that the nævus had originated n the parotid gland.

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14. *Adenoma mammæ; supervention of scirrhus.*

By J. Hutchinson, jun.

This case is of interest as illustrating the fact that an adenoma of the breast, after existing for many years, may serve as the centre for a scirrhous growth, and the family history presents some points which seem worthy of record. The facts are briefly as follows:

Mrs. H—, aged 54, came under Mr. Hutchinson's care for a tumour of the left breast which had been noticed six months, and which was then about three inches in diameter; this was in 1873. Some doubt was felt as to the diagnosis, but looking at the age of the patient it was thought best freely to excise the tumour. It proved to be an encapsuled adenoma, with a few small cysts in its substance. It only remains to be said that she has continued in good health, being last seen in December, 1886, when she was sixty-seven years of age.

Her daughter (the subject of the growth under consideration) noticed from girlhood a small round tumour just beneath the skin in the left breast; it was "as big as a cherry and did not grow." When aged 44, however, considerable growth occurred around the tumour, and when she came under the care of Mr. Hutchinson, senior, there was a hard mass adherent to the skin, the scirrhus having been nearly two years in growing. The affected part of the breast was freely excised, but not the whole gland, as the rest was quite sound. The axillary glands were affected with scirrhus and were removed. The patient remains well at the end of two years.

The specimen exhibited shows a rounded adenoma embedded in

scirrhus, which extends into the adjacent breast for the depth of about an inch to an inch and a half. The convex surface of the glandular tumour projects most markedly from the concave section of the scirrhus, and at first sight appears fairly isolated. On microscopic examination, however, the cancer is seen to have invaded the adenoma, which chiefly consists of fibrous tissue, the glandular structure being distinct but evidently degenerated. The scirrhus is of the small-celled variety, and the groups of cells in the adenoma are much more scattered than in the adjoining parts. Whether this implies that it really commenced in the tissue of the first tumour is doubtful.

Sir James Paget, in his 'Surgical Pathology,' p. 565, writes, "I am not aware of any facts which prove, what is commonly believed, that adenomata of the breast may become cancerous. Such things may happen, and on the whole one might expect that if a woman have a tumour of this kind in her breast, cancer would be more apt to affect it as a morbid piece of gland than to affect the healthy gland. But, I repeat, I know no facts to support this, and some that I have met with are against it."

It is very unwise to theorise from single cases, but the present case raises the suggestion whether it is not wisest to remove all adenomata of the breast, and whether in this particular case, if the adenoma had been removed earlier, any scirrhus would have developed. The parallel between epitheliomata developing around an old wart of the skin, and of melanosis originating from a congenital mole, is fairly close to the present case. [The specimen is in the R. C. S. Museum.] *November* 15*th*, 1887.

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15. *Villous carcinoma of the breast.*

By Bernard Pitts.

This specimen was removed from an unmarried lady, aged 53. She had been conscious of a lump in her breast for three months before she sought advice; the lump had increased from the size of a pea to that of a small walnut but had given the patient no inconvenience.

On examination I found a hard solid tumour situated on the

lower edge of the right breast, not fixed at its base, the skin not adherent, nipple not retracted, and no glandular enlargement in the axilla. From the age of the patient and the general feel of the tumour I had no hesitation in advising its immediate removal, and expected it would prove to be of a scirrhus nature.

The breast was removed on December 1st of last year. The wound healed by primary intention, and she was only confined to her bed for a week.

Directly the tumour was cut into after the operation it was apparent that the growth was of an exceptional character, and clearly not scirrhus; although the edge suggested a carcinoma yet the central portion of the growth became prominent on section, was soft, and almost melanotic in appearance, in fact resembled a melanotic sarcoma.

The prepared specimen shows an oval tumour, one inch by three quarters, surrounded by an imperfect capsule, which is closely connected with the breast-structure, and the tumour is in places infiltrating the gland. The dark staining is still manifest.

Mr. Shattock very kindly undertook the microscopical examination, and the following is his report:

"The tumour has an imperfect wall or capsule of fibrous tissue, from which arise branching processes very thickly invested with actively growing epithelium. So thick is this epithelial investment that in the undisturbed condition of parts the growth appears on section to be at first as intimately continuous in structure as a sarcoma or an ordinary carcinoma.

"In some places the nuclei of the deepest epithelium are oval in form and arranged vertically to the trabeculæ in which they lie. The cells are therefore of the columnar type, though the outline of the cell-protoplasm is not visible. In the rest of the epithelium the nuclei are spheroidal, very closely set, and equidistant.

"Some of the trabeculæ (the smaller of which are of extremely delicate, and lax connective tissue) are abundantly infiltrated with recent blood.

"The tumour must be classed as a villous carcinoma."

I can find in the 'Transactions' of the Society descriptions of three tumours of the breast which closely resemble the one I have ventured to bring before the Society this evening.

The first was described by Mr. Godlee in 1876, under the title of an "Anomalous Form of Blood-Cyst." The second by Mr. Pollard

in 1886, under the heading of "Duct Papilloma of the Breast;" and the third by Mr. Butlin as "A Recurrent Melanotic Sarcoma of the Breast." The specimen was referred to the Morbid Growths Committee, and they reported it as a "villous carcinoma."

On reading the descriptions of these cases and comparing them with the one now under consideration, one is struck by the many points of close resemblance both in the naked-eye and microscopic appearances.

In all there was a more or less perfect capsule, marked hæmorrhagic effusion, or staining of the growth. In each case there has been the same difficulty of obtaining a section owing to the foliaceous character of the growth and its tendency to fall to pieces, and the microscopical appearances have been very similar.

The clinical history of the cases has, however, been peculiar and difficult to explain.

Mr. Godlee's case was in a woman aged 67. The tumour was a recurrent one, and a gland in the axilla was infected with a similar growth.

In Mr. Butlin's case the patient was a woman aged 64, and there was a successive local recurrence of the disease but without any sign of implication of the glands, and, as Mr. Butlin stated, the clinical characters were throughout those of a sarcoma not of a carcinoma.

I do not myself recollect any case of carcinoma of the breast where recurrence took place on several occasions without any glandular or other manifestation. Possibly the clinical history of a villous carcinoma of the breast is quite different from that of an ordinary carcinoma. I shall endeavour to watch the progress of my patient and report if any local recurrence takes place.

March 6th, 1888.

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16. *Duct cancer of breast.* (*Card specimen.*)

By W. H. Battle.

Small tumour the size of a nutmeg embedded in the substance of the right breast removed by amputation of the entire breast on March 3rd, 1888.

The patient, a married lady aged 45, who had had several children, was under my treatment four years previously for cyst of thyroid; this has refilled. She was stout and well-nourished, has suffered from gall-stones, and the right axilla was occupied by a lipoma.

The tumour was firm in consistence, not separable from the breast substance, was situated to the upper and outer side of the nipple, painless and not tender. Skin not adherent, but apparently thinned over it; nipple not retracted. Had been noticed for about four months.

The axillary glands, which were enlarged, were removed at the operation, and on section presented well-marked evidence of new growth; they were not examined microscopically.

The growths were examined by Mr. Shattock, who has kindly given me the following report:

"*Histology.*—The tumour is composed of branched inter-communicating trabeculæ of connective tissue invested with epithelium. The epithelium is single-layered, in some places cubical, in others columnar, and it circumscribes laminæ of about the same breadth as the trabeculæ themselves. The spaces hold in many parts a faintly stained transparent, structureless, alveolated substance, in all respects like that seen in ovarian adenoma, and doubtless of the same kind, viz. mucus coagulated by the hardening fluids. At a spot where the section reaches the periphery there is displayed a well-marked capsule of dense connective tissue, and from this at one part arises the complex arborescent system of trabeculæ which form the tumour. The tumour would appear therefore to have arisen by papillary outgrowth from the wall of a cyst, and is to be classed as a villous carcinoma."

The naked-eye appearance of a recent section was more that of a vascular encapsuled sarcoma, there being none of the appearances characteristic of scirrhus tumour of the breast.

The wound healed at the end of ten days, and the patient left London a fortnight after the operation. *May 15th*, 1888.

17. *Secondary "duct-carcinoma" of the ribs.* (*Card specimen.*)

By Samuel G. Shattock.

As the subject of "duct-carcinoma" of the breast is one which needs further facts for its complete elucidation, the following may be worth recording, although it is an old museum specimen, C 216, in St. Thomas's Hospital Museum.

Portions of two upper ribs from the left side, each of which is the seat of a secondary new growth.

The larger tumour is 6 cm. in length, and has formed apparently (for part has been cut away) a fusiform swelling, projecting chiefly from the outer side of the rib. This growth presents no solid substance beyond the thin trabeculæ which circumscribe a multitude of cysts, some of them barely visible (where the growth appears more "solid"), others 2 cm. in diameter.

In the divided surface of the second tumour a lens discovers two or three branching trabeculæ ramifying in a substance which has a minutely porous texture, and in which are a few small, distinct cysts.

The bones were taken from a woman aged 60, whose left breast had been amputated a few weeks before her death by Mr. Joseph Henry Green.

It is recorded that the breast was believed generally to be scirrhous, but that some of those who saw it had doubts on the point.

Histology.—The tumour is constructed of mutually-adapted sinuous passages, irregularly dilated, sometimes in a degree to form cysts, but so freely communicating that the whole of the spaces probably constitute a continuous system. The walls of the spaces are of fibrous tissue, and their interior is lined with well-marked columnar epithelium. Here and there the relics of an osseous trabecula is to be seen lying in the cyst walls.

The histology of this specimen makes it evident that the tumour of the breast was of the kind known as duct cancer.

There are facts establishing the local recurrence of villous carcinoma of the breast (as in Mr. Butlin's case noticed in the preceding volume of the 'Transactions'); and glandular infection

has been observed by Mr. Godlee, the secondary growth being cystic and lined with columnar epithelium.

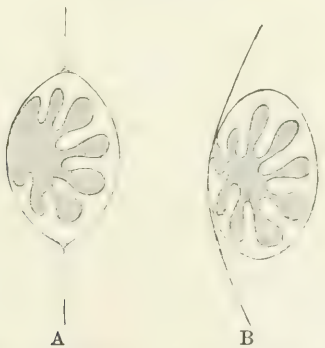
The present specimen shows that this kind of tumour may infect the bones, and that duct-carcinomas have all the characters typical of infective new formations.

The genesis of the secondary growths has still to be determined. The elements transported produce, probably, by multiplication, solid cylinders of cells which secondarily acquire a lumen, as is observed in the growth of thyroidal adenoma and other tumours, and as occurs in the normal process of gland-formation in the embryo.

Whether the secondary tumours are developed on the papillary type is another question requiring investigation, though such a mode of formation is difficult to hold.

And in the case of the primary tumour in the breast, it is not certain whether all such arise by papillary formation within a cyst —by *pro*cession—or whether some may not arise by the opposite method of *re*cession from a duct into the surrounding tissues (Woodcut 18).

WOODCUT 18.



Diagrams showing tumour formation: (A) by papillary outgrowth into a cyst; (B) by recession into the surrounding tissues, a capsule having formed around. The shaded part represents the cavity, the unshaded, the solid portion of the tumour; the straight and curvilinear lines represent ducts.

This difficulty obtains in other cases also, and it is chiefly by a careful study, in advanced formations, of the periphery that a conclusion can be formed. If there is a distinct cyst wall from

which the branching trabeculæ arise, and especially if there are considerable areas of the inner surface of the cyst wall free, the growth has arisen on the papillary type. The presence of free ends to the subdivided trabeculæ is not in itself enough, since the same appearance is presented in sections of tumours formed by recession. Should, however, a capsule form around a tumour produced by recession, it must become very difficult to determine on which type the growth has proceeded. The above diagrams will help to explain the question involved. *May 15th*, 1888.

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18. *Case of multiple cancerous tumours, many of them cystic, in the lungs, brain, bones, &c.; the primary tumour probably in the lung.*

By Joseph Coats, M.D., Glasgow.

[With Plate XX.]

The following case presents many features of interest, both in regard to its clinical and pathological aspects. For the clinical facts I am indebted to Prof. Gairdner, under whose care the patient was in the Western Infirmary, Glasgow. It will be observed that during life the symptoms pointed almost alone to the state of the brain and of the bones. After the *post-mortem* the notes were carefully scrutinized, and the nurse who had been in charge questioned, but without eliciting any facts which seemed to point to lung symptoms. This must be regarded as very remarkable if the view be correct that the primary tumour was in the lungs. The reasons in favour of this view will be given further on.

I. Summary of Clinical Facts.

Peter H—, aged 17, labourer, was admitted into the Western Infirmary (Ward 1) on September 8th, 1887. The most prominent symptoms on admission were complete amaurosis, with dilated pupils, with slight, almost questionable, external strabismus of

left eye, and a certain amount of nystagmus, but not any obvious paralysis of the oculo-motor (third) nerve on either side. His narrow forehead, &c., tended to throw into suspicion the state of his intelligence, but after most careful observation it was found that the higher cerebral functions were in no way interfered with by the disease. The early symptoms four months before admission had been regarded as gastric; vomiting and constipation, with pain in the frontal regions, being chiefly referred to. There was a suspicion, but only a bare suspicion, of a blow on the head having had something to do with it. No syphilis, and no previous constitutional disease of any kind, except rickets in early childhood, could be made out. There was no fever. The respiration was undisturbed. Pulse 84. There was considerable weakness of the lower limbs, and great emaciation, but no well-defined paralysis (perhaps relative paresis of left). The knee-jerk was sluggish on the left side, absent on the right.

Examination with the ophthalmoscope a week after admission showed engorgement of both discs, with a considerable amount of exudation extending beyond the disc, through which the vessels emerged at various points at some distance from the centre of the discs. The region of the macula was covered with minute white spots similar to those seen in albuminuric retinitis.

For some time after admission there appeared to be some improvement under the use of iodide of potassium, but on the whole it cannot be said that any essential change took place in the facts as above stated. Bodily weakness and emaciation continued to be marked features of the disease, so much so that, although cheerful and to a certain extent active in mind, he became entirely confined to bed. The knee-jerk disappeared entirely in the lower limbs, but there was still no distinctive paralysis.

On October 25th a painful swelling was reported in the right knee, and it had apparently been very insidiously developed, so as to suggest Charcot's disease. After consideration, however, this diagnosis was rejected on account of the limitation to one joint and the very considerable suffering. It was carefully treated, but without any appreciable result, and Dr. Gairdner began to suspect malignant disease of the osseous system. At a later period the appearance of other tumours on the bones gave additional force to this suspicion.

On November 27th an examination of the eyes showed the optic

nerves pale and prominent, and the vessels small. Outline of discs rather better defined than at previous examination, the affection being evidently of the nature of a papillitis with exudation rather than simple engorgement due to pressure.

On January 1st his temperature rose (for the first time) to 102·4°, and he complained of pain in the head. His sleep was disturbed, and there was an approximation to delirium. Emaciation was progressive. About this time osseous swelling was discovered in connection with the lumbar portion of the spine. There was considerable diarrhœa. On January 2nd and 3rd the urine (carefully examined throughout) was found to be for the first time distinctly albuminous, and with numerous hyaline casts and a few fatty ones.

A critical appreciation of all the facts was recorded with a view to clinical instruction on January 5th, but it contains nothing beyond what is mentioned above. The tumours in connection with the lumbar vertebræ had grown to a considerable size without any complaint of pain. Further tumours were now discovered, (1) to the right of the spinous processes in the dorsal region; (2) on the tenth left rib, outside the angle; (3) on the fifth right rib (softer than the others); (4) on the left femur; (5) in the upper part of the right femur (exceedingly hard, as also Nos. 2 and 4).

On January 12th a convulsion took place (for the first time), beginning in the left hand and extending to the left arm and left side of the face. The convulsion lasted twelve minutes. Conjugate deviation of the eyes and of the head to the left side was noted. There was no cyanosis, but complete unconsciousness. Four such convulsions took place within an hour and a half, after which the patient remained unconscious. Next day the fits had not recurred, but the patient was evidently dying, and he passed away in a state of profound coma.

II. Post-mortem Appearances.

So far as I can judge, the primary tumour in this case was in the right lung, and I shall therefore begin the description of the case with that organ.

The *right lung* was found firmly adherent. On cutting into this organ a large ragged cavity was discovered in the upper part of the lower lobe. The cavity measured an inch and a half in

diameter, and in some places its wall was formed by a grey tumour tissue which extended into the neighbouring lung tissue for a distance of about three eighths of an inch. The cavity contained a brownish pus. The neighbouring lung tissue was condensed, and it presented numerous dilated bronchi filled with pus.

In this lung there were, in addition, several small secondary tumours of a rounded form, which were present in the condensed area as well as in other parts of the lung. At the apex of this lung there was an old pigmented cicatrix with some pultaceous matter.

The *left lung* contained a single rounded tumour about an inch and a half in diameter, and of a mottled grey colour.

At the root of the lungs the bronchial glands were impacted into a somewhat solid tumour which was adherent to the bifurcation of the trachea and produced a bulging into both bronchi. At two places in the wall of the right bronchus there was a white area visible internally, as if the tumour had grown from the glands through the bronchial wall.

The *bones* presented numerous secondary tumours; several of these were in the ribs. On the right side the fifth rib presented an elongated soft swelling, just outside the junction of the osseous and cartilaginous portions. There were similar swellings on the fifth, sixth, and tenth ribs on the left side, all of them in the axillary line. These tumours evidently replaced the osseous tissue to a large extent with soft tumour tissue. The *right femur* had bulky tumours in the cancellated tissue at both extremities, and there were fractures of the bone in both of these situations. A section through these shows that the lower end is distended by a bulky tumour which for the most part replaces the cancellated tissue. The bone is fractured three and a half inches above the condyles. The upper end is also occupied by tumour, to the extent of about four inches of the shaft, and there is a fracture just beneath the great trochanter. The *left femur* has also a tumour about the junction of its lower and middle thirds, which forms a bulging inwards about two and a quarter inches in long diameter.

The *vertebræ* showed tumours projecting usually from their lateral aspects. The sixth and seventh dorsal vertebræ presented rounded swellings on their right lateral aspects which involved the heads of the ribs, perhaps more than the bodies of the vertebræ. There was also a tumour on the right side of the first lumbar vertebra, and

the body of the fourth lumbar vertebra was almost entirely occupied by a tumour which projected on either side and in front. The *wing of the left ilium* was occupied by a tumour bulged inwards, close to the crest, and involving about half of the wing. There was a smaller tumour immediately beneath the anterior superior spine of this bone.

The brain.—This organ contained at least twenty-two separate tumours, mostly cystic, and distributed in almost every region. In removing this organ it was found that one of the tumours communicated with the surface, and had formed a slight adhesion to the dura mater, so that it was torn during removal. Some gelatinous fluid escaped. The following description of the various tumours was obtained by hardening the brain, and dividing it horizontally at four different levels. Doubtless some smaller cysts escaped notice. The tumour already mentioned as having been torn into is the only one having any approach to solid tissue. Its tissue is very soft, and contains many developing cysts. The others were pure cysts containing for the most part a brown glairy fluid, and lined by a delicate membrane, which under the microscope was seen to consist of a layer of large flat cells.

Right hemisphere.—(1) A large cyst of a rounded or slightly quadrilateral shape, and about one and half inches in diameter, lying with its summit close to the surface near the vertex of the hemisphere, where it undermines the upper part of the ascending parietal convolution. The floor of the cyst is about the level of the lateral ventricle, but it lies outside the latter. Its depth from above downwards is about one inch. Its internal border is about half an inch from the longitudinal fissure and its external border about one eighth of an inch from the lateral aspect of the hemisphere. (2) Immediately behind this there is a small cyst lying about an inch from the longitudinal fissure. It is somewhat elongated, ascending alongside the upper border of the large cyst. (3) A cyst, half an inch in diameter, lying in front of No. 1, with a space of five eighths of an inch between their proximate borders. (4) The tumour already referred to as having been torn. It is an irregular semisolid structure situated in the occipital lobe. It has the appearance of continuing backwards the lateral ventricle, but on careful examination it is seen that a thin lamella of brain tissue always separates the tumour from the ventricle. The tumour is elongated and has a diameter from before

backwards of two inches. It comes close up to the internal border of the hemisphere, and approaches to the external border as near as a quarter of an inch in some places. (5) In the parietal lobe near the external surface and at the level of the lateral ventricle a small cyst. (6) In the frontal lobe in contact with the median surface of the hemisphere, and about half an inch from the anterior extremity, a small cyst three eighths of an inch diameter. (7) About the same level as the last, but in contact with the external surface of the hemisphere, a very small cyst, half the diameter of the last mentioned. (8) In the frontal lobe, at its extreme anterior extremity, almost in contact with the surface, and just beneath the level of the last two, a larger cyst about five eighths of an inch in diameter. This contains a brown coagulum, which under the microscope only shows débris. (9) Among the superficial convolutions just outside the island of Reil a cyst about a quarter of an inch in diameter. It is immediately in contact with the fissure of Sylvius. (10) and (11) Two small cysts can be felt in the temporal lobe, near the anterior extremity and just beneath the inferior surface.

Left hemisphere.—(1) A large cyst occupying much the same position as No. 1 in the right hemisphere, but rather larger. It has a diameter from before backwards of two and one eighth inches. Its internal border is only a quarter of an inch from the median surface, and the external border trends rather outwards towards its middle, giving a transverse diameter at its widest part of one and five eighths of an inch. (2) Immediately in front of the preceding a small cyst which lies close to the internal border of the hemisphere. (3) In the frontal lobe and slightly below the level of No. 1, a cyst about half an inch in diameter and of an irregularly triangular shape. It is close to the anterior border of the hemisphere, separated only by the thickness of the grey matter of a convolution, and it is also close to the median surface. In its lower part the cyst passes rather backward and outward and is closely connected with the next. (4) In the frontal lobe, close to the anterior border of the hemisphere, and touching the median surface, a cyst about half an inch in diameter, which just faces No. 6 of the right hemisphere. (5) A cyst half an inch in diameter just outside the floor of No. 1. It corresponds in level with the posterior part of the thalamus opticus. (6) In the occipital lobe a larger cyst somewhat corresponding with No. 4, in the right hemisphere, about three quarters of an inch in

diameter, immediately behind the preceding cyst, and just outside the posterior cornu of the lateral ventricle. (7) and (8) Two small cysts in the occipital lobe superficial to the preceding one and close to the superior surface of the brain. (9) A small cyst in the middle of the island of Reil and in contact with the surface. (10) In the convolution over the island of Reil an oval cyst half an inch in diameter and in contact with the external surface of the hemisphere.

Cerebellum.—A cyst of considerable size in the extreme posterior part of the left lobe, causing a considerable bulging backwards.

The *pancreas* showed at its extreme termination a small cyst containing yellow material, which appeared to infiltrate the neighbouring portion of the capsule of the left kidney. The kidneys were otherwise normal.

The *liver* contained a few white tumours of small size, none of them more than one eighth of an inch in diameter. The mesentery and small intestine presented on their parietal surface numerous small white nodules, and there were also some larger tumours which may be enlarged lymphatic glands. In the lower part of the small intestine the mucous membrane was thickened and of a grey colour. The large intestine was contracted and in parts pigmented.

III. Microscopic Characters.

In all the situations where solid tissue existed the tumour showed what may be described as a typically cancerous structure but with a remarkable tendency to the formation of cysts, apparently by metamorphosis of the cancerous tissue.

The *lungs* presented probably least of the cystic tendency. In a section of the advancing edge of tumour tissue in the wall of the large cavity in the right lung, the tissue, as shown in Plate XX, fig. 1, was very regular in structure. There are spaces resembling in size the lung alveoli, and with delicate walls like those of the alveoli. The spaces are filled with epithelial cells. The peripheral cells are usually cylindrical, and there is frequently a regular layer of cylindrical cells lining the spaces. The remaining cells are somewhat irregular in shape. In this situation there is very little of the cystic character. Around this advancing edge of the tumour the lung tissue is the seat of considerable hæmorrhage. The lung alveoli are full of blood, and there are other signs of irritation in

DESCRIPTION OF PLATE XX.

In illustration of Dr. Joseph Coats' paper on Multiple Cancer of Lungs, Bones, Brain, &c. (Page 326.)

FIG. 1.—From supposed primary tumour in right lung. Alveoli about the size of normal lung alveoli, filled with epithelium, which is columnar at the peripheral parts. × 70.

FIG. 2.—Advance of cancer in perivascular lymphatics; *a*, a branch of pulmonary artery, *b b*, cancerous formations occupying the lymphatics in the sheath of the vessel. From supposed primary tumour. × 70.

FIG. 3.—From secondary tumour in lung, the epithelial masses irregular in form, but lined with columnar epithelium. *a a*. Partial cystic formation. × 70.

FIG. 4.—From tumour in brain. *a a*. Small developing cysts lined with cylindrical epithelium. *b b*. Larger cysts with swollen irregular epithelium. × 70.

FIG. 5.—From tumour in femur, cysts encroaching on bone. *a a*. Spiculum of bone. *b b b*. Portions of three cysts lined with cylindrical epithelium (goblet-cells) and impinging on the bone. × 70.

the presence of epithelial cells, irregular in arrangement, in the midst of the blood.

It cannot be said that the tumour is advancing to any considerable extent by the penetration of the growing epithelium into the lung alveoli and using the walls as a stroma, although the size of the spaces in the cancerous structure suggests this. There are indeed places where the cancerous epithelium, distinguished by its tendency to the cylindrical form, appears to be growing in alveoli, but this is nowhere unequivocal, and, for the most part, the alveoli, with the blood and catarrhal cells in them, are being pushed aside.

There can be no doubt, on the other hand, that the tumour is advancing by the lymphatics. It is not uncommon to meet with appearances, such as those represented in fig. 2, in which the lymphatics around a branch of the pulmonary artery are filled with epithelium having the characters of that of the tumour proper.

In the case of the more distinctly secondary tumours in the lung the structure is somewhat similar to that just described. The alveoli are much less regular, however, and they seem to run into one another more. There are still the cylindrical cells lining the spaces, but the appearances are those of a more vigorous growth than in the other. There is here also more distinct evidence of cystic formation (see fig. 3). The central parts of the alveoli are occasionally occupied by a clear colloid material, in which there are sometimes large swollen cells. This condition, however, is very inconsiderable compared with what exists in the bones and brain.

Sections of the *bronchial glands* showed an appearance not unlike that of scirrhous cancer. The glands are obviously indurated and largely replaced by dense connective tissue. In the midst of this there are collections of cells, which are frequently in the form of elongated processes, such as we see so frequently in scirrhus. I suspect that this modification of structure is due to the fact that the glands were indurated before the occurrence of the cancer. There were evidences of an old tuberculosis at the apex of the right lung, which may account for the induration.

The condition of the *bones* was examined by decalcifying a portion of the expanded lower end of the femur and making sections after embedding in celloidin. The structure here is very largely cystic. There are wide spaces filled with colloid material, in which a few irregular cells are present. These spaces sometimes show a regular

lining of cylindrical cells (see fig. 5), but not infrequently this is interrupted at intervals or absent. There are places where the spaces are small, and where there is a regular layer of cylindrical cells with a calibre containing colloid matter. There are even parts, especially near the surface, where the collections of cells are elongated and almost devoid of any cystic tendency. Some spiculæ of bone remain, but they are evidently undergoing atrophy before the advancing tumour tissue (see fig. 5).

The tumour in the *brain*, already described as the only one which presented any solid tissue, was very soft and consisted of little more than a congeries of cysts. Sections of it were made after embedding in celloidin. The structure is like that in the bones, namely, irregular spaces with colloid contents, occasionally with a layer of cylindrical cells lining them (see fig. 4). These cells are very often swollen up, presenting the characters of goblet-cells and reminding one of the appearances often seen in colloid cystoma of the ovary. They sometimes formed a single layer lining a small cavity (as at *a* in fig. 4), while in other places (as at *b*) they were more irregular.

IV. Remarks.

We have here a case in which, with a large ulcerating tumour in the right lung, there were numerous secondary tumours in lungs, bones, and brain, and a few tumours also in the liver, pancreas, and peritoneum. In the bones and brain the tumours showed a remarkably cystic character, while this was only slightly developed in the tumours in the lung. The tumours, wherever they show sufficient solid tissue, have the characters of cylinder-celled epithelioma, and in some places the cancerous structure is typical.

Without presuming to any dogmatic statement, I may express my belief that the tumour in the right lung is the primary one. This tumour was obviously old, as it was in the form of a large ulcerated cavity with cancerous walls. Besides, it is the only tumour, so far as observed, which had led to cancerous infection of the lymphatic glands. The bronchial glands were impacted and were the seat of cancerous growth. I believe this affection of the glands to have been comparatively old, especially as the disease had advanced to the wall of the contiguous bronchial tube. Tumour tissue had begun to show itself inside the bronchus in the form of two white nodules.

So far as this tumour is concerned it conforms to the description of recorded cases of primary cancer of the lung. Eberth[1] describes a case in which the lower lobe of the lung was occupied by a large medullary mass. The marginal parts of the tumour showed what appeared to be lung alveoli occupied by cylindrical epithelium. The structure of the tumour is compared with that of malignant adenoma of the stomach or intestine, the form of tumour usually designated cylinder-celled epithelioma.

Langhans[2] goes fully into the development of primary cancer of the lung. He derives it from the mucous glands, and in accordance with that the epithelium is cylindrical.

Perhaps the most completely recorded case of primary cancer of the lung is that of Finlay and Parker.[3] In this case there was a large soft mass in the upper part of the lower lobe of the left lung. Besides this primary tumour there were a number of secondary ones in both lungs and a few in the liver. The bronchial glands were enlarged and cancerous. In structure this tumour was a typical cylinder-celled epithelioma. The authors describe the growing epithelium as to some extent inside the lung alveoli.

In Finlay and Parker's paper there is reference to several other recorded cases of primary cylinder-celled epithelioma of the lung. One of these, namely, that of Machiafava,[4] has some analogies with the case here recorded. Besides the primary tumour in the lung there were numerous nodules in the brain, varying in size from a millet-seed to a nut. There were also secondary tumours in the lungs, and there were tumours in the frontal bone, which are said to have sprung from the periosteum. All the tumours had cylindrical epithelium.

I have myself recently encountered a case of primary cancer of the lung in which the structure was that of the cylinder-celled epithelioma. In it I was able to observe not only the mode of advance by the lymphatics, similar to that shown in fig. 2, but also an extension into the lung alveoli. The tumour, which was a bulky soft mass, had caused irritation in its neighbourhood, the result being that the lung alveoli contained numerous large catarrhal cells. In many places it could be seen that the cancerous

[1] 'Virchow's Archiv,' vol. xlix.

[2] Langhans, 'Virchow's Archiv,' vol. liii, p. 470.

[3] Finlay and Parker, 'Med.-Chir. Trans.,' vol. lx, p. 313.

[4] Machiafava, 'Rivista clinica di Bologna,' May, 1874.

epithelium was present in the alveoli along with the catarrhal cells. This was the more readily detected as the catarrhal cells were very slightly stained with alum-carmine, while the cancerous cells were deeply stained.

So far as I can find there is no case on record in which such an extension to the brain and bones has occurred as that observed in the case which is the subject of this paper. It might possibly be suggested that these numerous cysts in the brain had some other origin to that assumed here, that they might, for instance, have been of congenital origin or otherwise. The existence of cystic tumours in the bones, however, entirely disposes of this objection as these were developed under observation and were obviously secondary new formations.

The process of formation of the cysts was apparently by a kind of mucous or colloid secretion of the epithelium very similar to that which one sees in ovarian cysts. It is very unusual to have such a transformation in cancerous tumours, and it is perhaps all the more remarkable from the fact that there was very little or none of it in the primary tumour. It is difficult to understand how a tendency such as this should have manifested itself to such an extraordinary extent in the bones and brain, while even in the secondary tumours in the lungs it was very subordinate, although present to a slight extent as shown in fig. 3.

The mere existence of this tendency we may, perhaps, trace to the seat of origin of the cancer. According to Langhans—and most authors seem to agree with him—the tumour takes origin in the mucous glands of the bronchi. If this be so then we should be prepared for a certain amount of mucous or colloid secretion, such as we often find in the similar tumour in the intestine. It seems as if the bones and the brain were specially favorable seats for the exaggerated development of this tendency.

May 1*st*, 1888.

19. *Cystic carcinoma of neck; removal; recurrence.*

By Charters J. Symonds.

Alfred T—, aged 56, admitted into Guy's Hospital, August 29th, 1883. Eight weeks ago noticed a hard lump under right ramus of his jaw. There was a tumour on right side extending from the angle of the jaw to a little below upper border of thyroid and from sterno-mastoid to half an inch across the left of middle line. The skin was somewhat movable over it and the tumour was partly movable over the deeper structures. The outline was well defined. The skin was adherent over the lower part, and the growth was closely connected with the thyroid cartilage. The tongue can be protruded to the full extent, and no ulcer could be discovered within the mouth nor by laryngoscopic examination in the pharynx or larynx. None of the nerves were compressed. The tumour was taken to be a sarcoma growing from the deep fascia of the neck. The man suffered a good deal of pain.

On September 7th the growth was removed. Two incisions were made from the angle of the jaw to near the tip of the sternum, the included piece of skin removed with the growth. The enucleation was commenced from below, and everywhere the growth was found closely adherent. It proved to be a large cyst with thick white walls. The finger was introduced, and using this as a guide the tumour was extirpated in doing which parts of the anterior bellies of both digastrics, the posterior of the right, the stylo-hyoid, part of omo-hyoid, and the thyro-hyoid and sterno-hyoid muscles were removed. The submaxillary gland was in part removed. About one inch of the internal jugular vein was resected, being infiltrated with growth, but the carotid with the nerves was free. Several arteries, including the lingual, ascending pharyngeal and facial were torsed. The thyro-hyoid and crico-thyroid membranes were exposed, there being much risk of penetrating the structure. One gland was also removed, being infiltrated. The pharynx was exposed, and could be seen to move in and out in respiration. The hypoglossal and descendens noni with the sympathetic were exposed but left uninjured.

The wall of the cyst was five eighths of an inch thick. The

wound was closed by wire, a large drainage-tube introduced. The whole operation lasted two hours. Five hours later his pulse was 100, and his temperature 98·2°. The spray was used throughout.

On the sixth day the sutures and tube were removed.

On the eleventh day he was allowed to get up, and on the twenty-first was discharged.

On the sixth day it was noticed that he did not protrude the tongue freely, that it pointed to the right side, and that this half was also a little wrinkled and flabby. This condition remained on his discharge and evidently indicated that the hypoglossal had been injured. This was probably done when the hyoglossus was divided. Rapid recurrence took place, and on November 10th (four and a half weeks from the time of operation) he was re-admitted. There was then a large mass beneath the chin infiltrating the floor of the mouth, and the cicatrix was closely adherent to the larynx. There were two fungating buttons in the line of old incision. A mass also extended downwards under sterno-mastoid, and the tongue was partly fixed. It was evidently hopeless to attempt removal. The tongue was still wrinkled and softer on the right side, and protruded to the right.

The wall of the cyst showed all the characters of squamous epithelioma, and resembled in structure exactly the case recorded by Mr. Treves in the last volume of the 'Transactions.'

In that paper will be found a discussion of the probable origin of these tumours. *January 17th*, 1888.

VIII. DISEASES, ETC., OF THE DUCTLESS GLANDS.

1. *Hypertrophy of an accessory thyroid in a case of spindle-celled sarcoma of the thyroid.* (*Card specimen.*)

By Samuel G. Shattock.

Henry S—, aged 42, coachman, admitted into St. Thomas's Hospital under Mr. Sydney Jones, June 30th, 1887. There was a large tumour on the right side of the neck, extending from the lobule of the ear to the clavicle, anteriorly almost to the middle line, posteriorly rather beyond a line drawn vertically downwards from the mastoid process. The tumour rises and falls during deglutition and was first noticed about five years ago. On the left side of the neck is a soft diffuse swelling, over which the superficial veins are dilated; this is separated from the tumour first referred to by a distinct sulcus. The bronchocele is soft and about the size of half an orange.

July 7th.—Mr. Jones removed the tumour of the right side, a ligature being passed around the connection between the tumour and the other half of the thyroid. The patient was discharged with the wound not quite healed August 6th, 1887.

On October 22nd, 1887, he was readmitted. The wound had never quite healed, and there was a tumour the size of an orange over the right side of the larynx.

November 2nd.—Mr. Battle removed the growth, which was found to invade the sheath of the carotid and the œsophagus.

15th.—Wound nearly healed and patient comparatively well. Dr. Semon found, on laryngoscopic examination, that there was a distinct inroad of new growth into the right side of the pharynx reaching up to and including the right aryepiglottic fold.

Recurrence was observed in December, and on the 30th trache-

otomy was performed under chloroform, a large mass of the tumour being removed together with what was supposed to be a diseased lymphatic gland.

On February 22nd the greater part of the tumour (which had again recurred) was enucleated. A similar operation was performed on March 28th. The common carotid was tied by Mr. Battle on April 5th, 1888, to stop secondary hæmorrhage.

Death occurred on April 7th.

Examination of the parts shows a large mass of sloughing tumour-substance occupying the right side of the neck.

The new growth passes posteriorly beyond the middle line and meets the left lobe of the thyroid, which is, however, easily separable from it by dissection. The pharynx and œsophagus are displaced towards the left and partly filled, partly compressed, by the tumour; the common carotid passes into its substance. Anteriorly the growth crosses the mid-line and invades the adjoining part of the left lobe of the thyroid.

In regard to the left lobe itself the upper, lower, outer, and posterior limits are readily defined by dissection; the lobe measures two and a half inches in length, is lobulated externally, and its section presents, to the naked eye, the normal structure. Its summit is constituted by an isolable mass of thyroid tissue, 2·3 by 1·2 cm., which admits of being clearly enucleated from beneath the capsule of the gland. Posteriorly there is a second isolable, somewhat smaller lobule, situated also beneath the general capsule. There were no secondary growths in any of the internal organs.

The tumour retained throughout its course the microscopical characters of a typical spindle-celled sarcoma, and the only pathological fact worth recording in connection with the case is, that the supposed lymphatic gland removed with part of the recurrent tumour on December 30th, 1887, consists throughout of exquisite thyroidal tissue; it measures 2 by 1·3 cm. in its chief diameters, is wholly isolated from the new growth, and is obviously either an accessory thyroid in process of hypertrophy or, what is equally interesting, an adenomatous growth in connection with the remains of the gland similar to that present in the apex of the opposite lobe. There is no reason for supposing such isolated masses of thyroid tissue to be less functional when within the gland capsule than when in the form of accessory portions beyond it.

Horsley has shown by direct experiment that if half of the thyroid be removed the rest of the gland undergoes proportional hypertrophy.

The overgrowth of an accessory thyroid under such circumstances as the present is recognised as of considerable interest in relation to the absence of myxœdema after destruction of the main gland by disease or its removal by operation. And it is worth careful investigation whether, in cases of complete destruction of the renal capsules without the signs of Addison's disease, the function of the glands is not in some degree maintained by the hypertrophy of accessory adrenals. *April 3rd*, 1888.

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2. *Cyst-adenoma of thyroid.* (*Card specimen.*)

By J. H. Targett, M.S., for Dr. Farrant Fry (Swansea).

History.—Thomas L—, aged 63, always enjoyed good health. Two months before admission noticed a swelling on the right side of the neck which has slowly increased. Latterly his voice has been thick and he has lost flesh. The right half of the thyroid is now much enlarged and very hard. It was freely excised and the patient soon recovered. The subsequent history shows that the tumour recurred *in situ* within three months of operation, causing great interference with deglutition and rapid loss of flesh.

Examination of tumour.—On section, the tumour appeared to consist of tough fibrous tissue in which a great number of cysts were embedded. These were of all sizes up to that of a pea, and were filled with soft white material. In some places this had fallen out, leaving smooth-walled cavities. Only a portion of the tumour was preserved but this was not encapsuled.

On microscopic examination, many of the cysts were found to contain papillary ingrowths covered with cylindrical epithelium, and these completely filled the cavities. In some spots they had undergone colloid degeneration. The basis of these ingrowths consisted of delicate connective tissue containing capillary vessels.

The structure of the tumour thus corresponds with that of cyst-adenoma proliferans (Wölfler), which is apt to recur, as in the present instance. An apparently similar tumour is described in 'Path. Trans.,' vol. xxxvii, p. 507. *March 20th*, 1888.

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3. *Cancer of the thyroid isthmus.* (*Card specimen.*)

By R. G. Hebb, M.D.

Robert A—, aged 41, was admitted to the Westminster Hospital under the care of Dr. Allchin, February 21st, 1887, for paraplegia. There was angular curvature of the spine in the upper dorsal region. The onset of the symptoms was gradual and began with pain and weakness in the right thigh about two years before admission. During his illness had emaciated greatly. Personal and family history immaterial.

Post-mortem examination forty-four hours after death.—Body much emaciated; several bedsores. Cancerous growth round about and involving the bodies of the second, third, and fourth dorsal vertebræ. By invasion of the canal the cord is pressed on at this point and the dorsal region much softened. There were no enlarged glands in the thorax or abdomen, but there were numerous secondary growths in the lungs and kidneys. The thyroid isthmus is changed to a tumour about the size of a hen's egg. It is cystic and enveloped in a tough fibrous husk, especially the lower half, while the upper half is more fleshy; the internal aspect of the capsule is irregular from projecting nodules. Owing to the pressure of the tumour the trachea is bulged inwards. The lateral lobes of the thyroid are normal.

Microscopical examination of the primary growth and of the secondary nodules show that they are made up in great measure of closed follicles lined by epithelium, the contents of the tubules being "colloid" matter. The type of the neoplasm is essentially that of the thyroid. (In some secondary growths the tubules are so closely compressed that the opposing epithelia are in contact; in others there is marked fibrosis at the centre.)

May 15th, 1888.

4. *Case of Addison's disease.*

By HARRINGTON SAINSBURY, M.D.

THE case from which the specimens now shown were taken ran a typical course; for this reason it will not be necessary to give more than the briefest records.

Daniel G—, a police sergeant, aged 38, was admitted into the Royal Free Hospital on August 16th of this year. He was admitted for great weakness with well-marked emaciation; for darkening of the skin, especially noticed about face and backs of hands; for abdominal pain with frequent vomiting, the vomit occasionally containing blood; for extreme feebleness of pulse.

The man gave a healthy history up till 1878, when, in the performance of his duty, he was severely kicked about the abdomen and was obliged to give up duty for fourteen days. Since that time he had suffered occasionally from sharp pain in the abdomen and sickness but had been able to keep at his work. On October 11th, 1886, again in the discharge of his duty, he was very severely kicked about the abdomen and chest, and he also received damage in the testes. He vomited at the time and continued vomiting all night off and on. On the 12th he was sufficiently recovered to be able to give evidence, but on the 13th he was obliged to give up his duty, and has never been able to return to it.

The symptoms described on admission had gradually appeared and progressed, and amongst others there was much shortness of breath on exertion.

I may mention that in the family there was no definite history of illness except of rheumatic fever.

To the present state on admission already given I need only add a few details. There was some pigmentation on the mucous membrane of the lips; the backs of the hands, in addition to the general deepening of tint, showed small dark specks of pin-head size, they also presented a curiously polished appearance; the abdomen and thorax showed scattered spots of brown pigmentation; a large patch of a much deeper brown on the abdomen marked the site of past irritation by iodine.

The abdominal pain mentioned was referred to epigastrium and left hypochrondrium but it was not limited to these parts, the lumbar

regions and particularly the right lumbar region being much complained of.

In the course of the case the emaciation increased, and the prostration became extreme.

The patient died on September 19th. During the last three days he was more or less delirious, sometimes wildly so; he appeared also to be in much pain.

Whilst in hospital, the sickness, which was a constant symptom, frequently showed blood in the vomit.

The patient's colour varied a good deal in depth of tint during the progress of the case, but at no time was it very intense.

At the *post-mortem* the following appearances were noted:

The subcutaneous fat tolerably preserved. In the abdomen a few old adhesions in the region of the spleen; the hepatic flexure of the colon more adherent than usual to the parts posteriorly; between the liver and right suprarenal old and somewhat dense adhesions. Search was made for any evidence of past traumatism; none was found. The abdominal organs, spleen, kidneys, liver, were healthy looking. A few ecchymoses marked the mucous surface of the stomach, small and large intestines presented nothing abnormal. No disease of the spine.

Right suprarenal capsule much enlarged, converted into a dense fibro-caseous mass (*vide* specimen). At one or two spots there were foci containing a puriform fluid, evidently the result of softening of the cheesy material.

Left suprarenal capsule (before removal an appearance as of a superficial scar was noted in the position which the capsule should occupy): No enlargement of the body to be felt; accordingly the mass of fat situated at the head of the left kidney was removed for further examination. On section through this fat a minute fibrous centre of triangular form was seen. Dissection from without inwards was then attempted but was given up for what appeared a much safer method, viz. a series of vertical sections. These showed the capsule embedded in the fat and reduced to a minimum. To the naked eye no trace of the normal structure remained; there was nothing but a fibrous mass which by its shape and position alone declared its nature. One section is of special interest, for it not only shows more clearly the shape of the capsule but also the presence of a small caseous nodule with central breaking down. Another section shows another small caseous focus.

I may add in completion of the *post-mortem* record that, with the exception of scarring at the apices of the lungs and the presence there of one or two minute caseous masses, no fibro-caseous changes anywhere else were discovered.

There are several points in this case to which I am desirous of drawing attention. I may first, however, state that there can be no question as to the nature of the case. Both clinically and pathologically it stands as typical of Addison's disease.

The first point concerns the etiology. Traumatism plays here so distinct a part in the clinical history that I think it must be accepted as the determining cause. Dr. Greenhow gives a few cases in which traumatism was probably the cause, but in none, I think, is the evidence quite so strong as in this case. I would moreover point out that the injuries involved serious and extensive irritation of the sympathetic system.

The next point concerns the nature of the pathological changes in the capsules. As to the right capsule the condition found there needs no discussion,—it will be admitted as typical of the common form of disease. As to the left capsule there can, I think, be no doubt that the condition found is identical in nature with that in the right, —the fibroid change, the caseous focus breaking down at its centre these give sufficient evidence; but I would draw special attention to the extreme to which degeneration and of shrinkage has here proceeded, and I would ask whether we are not to consider that in process of time the right capsule might have undergone a similar reduction in bulk. This question of the extreme to which fibroid shrinkage and disintegration may proceed is of interest not only from the point of view of morbid histology but also from the point of view of the pathology of Addison's disease, with special regard to the cases, more recently described, of this disease with atrophy of the capsules. If the replacement of the tissues of the adrenal by fibrous tissue may be as complete as it frequently is in Addison's disease, and if this fibrous tissue may then suffer reduction to the extent here witnessed, may not the process advance yet a step further and end practically in the complete effacement of the organ? (In the present case it is, I think, clear that the left capsule would have shrunk yet further had the patient lived.) If this be so it is more than probable that some of the cases of atrophy of the capsules which have been held to disprove the fibrous-caseous theory of Addison's disease will really be included by that theory

and will themselves represent ultimate stages of the fibro-caseous change. Is it impossible too that some of the cases of reported absence of one of the capsules *e. g.* in Averbeck's Tables (Table I, Case 45), or even of both capsules (Dr. Kent Spender's case), may really be cases of extreme atrophy which have, however, passed through a fibro-caseous stage?

One last point in connection with this case. In the patient's history there are two distinct traumatic records, and at the *post-mortem* we find the capsules in two distinct stages. I will not venture beyond this.

In conclusion, I would suggest that in all cases of apparent atrophy of a capsule the safest method of procedure in searching for it is by a series of cross sections as here.

November 1*st*, 1887.

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5. *Case of Addison's disease in a boy of* 17*; of eight months' duration; bronzing seven months'; twitchings. Death. Capsules fibro-caseous; chronic tuberculosis round them, and disseminated thence over peritoneum.*

By Samuel West, M.D.

Charles A—, single, aged 17, a waiter at a restaurant, was admitted into the Royal Free Hospital on June 16th, 1887, with Addison's disease.

He was the youngest of four children, who were all healthy, as he had been until five months ago, when he caught cold and had pain in the right side with some cough. He left work and was laid up for three months, when he went to a Convalescent Home for a month but returned no better, and shortly after came to the hospital, finding himself unable to work. During all this time he had been steadily growing weaker without any evident cause. The appetite had been extremely bad, and he had lost flesh. Vomiting had occasionally troubled him, but it had not been severe. The colour had been noticed to be darker than natural to him in March, *i. e.* about a month after the commencement of his illness.

The patient's mother died young, but from what cause is not known; there was a history of phthisis in her family, and one paternal uncle died also of phthisis.

The patient was very thin and feeble, with cold extremities and a feeble pulse, but there were no physical signs except the bronzing.

The pigmentation was general over the whole body, but most marked on the neck and face, and on the lower part of the abdomen and upper part of the thighs. In these latter places the skin was very dark and mottled. Irregularly scattered over the whole body were small black spots not raised above the skin; they were especially numerous on the face, but some of them, the patient said, were of old date, though many he knew had come lately. On the face the bronzing was uniform, with many small black spots; irregular patches of dark pigment marked the line of junction of the lips. A small patch of pigment was found on the mucous membrane of the lip just above the left upper incisor, and other patches existed on the middle of the inside of both cheeks. The whole neck was deeply and uniformly bronzed. The arms were less pigmented than the rest of the body; there was a marked absence of pigment in the folds of the elbows and axilla, but there were many black spots which were more conspicuous from the comparative lightness of the skin round them. On the back the bronzing was uniform, except for transverse lines of deep brown colour corresponding with the lower dorsal spines. The thighs were deeply pigmented, as described, and mottled, but below the knee there was but little excess of pigment. The nipples were very dark. The retinæ showed no trace of abnormal pigmentation.

There was almost complete loss of appetite but the tongue was clean. Vomiting occurred occasionally but without any fixed relation to food. The urine was of low specific gravity, 1012, but otherwise normal.

Up to August 9th the patient slowly improved. The temperature was generally normal but rose once to 100°. The only change which occurred during this time was the development of several new patches of pigment.

On June 30th, a new patch was found on the right border of the tongue.

On July 2nd, a pale brown triangular mark developed on the left side of the root of the nose, and became very distinct in the course of a few days.

On the 9th, several more black spots were discovered on the trunk, arms, and face.

On August 9th the patient, who had been for some days well enough to get up and go into the square, was suddenly seized with vomiting and severe pains in the right shoulder and in the right hypochondriac and lumbar regions. This attack completely prostrated him ; he became alarmingly weak and lost all appetite. The abdominal muscles were kept rigid, and great tenderness was felt in the epigastrium ; the colour of the skin seemed to become deeper, but it varied much on different days according as it appeared to the patient's general condition.

On the 16th the temperature, which had hitherto been normal, rose to 102°, and remained at this height for the next few days, during which time the tongue became very dry, the vomiting continued and the appetite failed entirely.

On the 23rd, the weakness was extreme. The pain in the lower part of the abdomen was better, having been relieved by rubbing with extract of belladonna. For the last few days there had been some twitching of the face and limbs.

On the 28th, the patient became very faint, and passed into a condition of unconsciousness for about five hours, when he had an attack of convulsions in which he threw himself about and could not be kept in bed. The right side of the face twitched more than the left. These twitchings lasted all night.

In the afternoon of the next day he began to throw himself about as if in unbearable pain, and he complained of great agony in the right hip, but no cause there for the suffering could be found.

On the 30th, the vomiting became very severe and no urine was passed at all for twenty-four hours.

From this time till his death on September 21st the patient lay so weak as to appear almost unconscious, vomiting constantly, and having positive repulsion for food, so that for several days the only food he took was two or three oysters.

On the 21st, muscular twitchings returned, and shortly after the lad became unconscious and died.

Post-mortem examination.—The body was small and ill developed for the age, presenting the appearance of a lad of thirteen or fourteen only. The pigmentation was as described, but paler in colour. The right pleural cavity was completely obliterated, and also the anterior part of the pericardium.

The liver was completely adherent to the diaphragm, and in these adhesions as well as in the capsule of the liver were very numerous grey and yellow tubercles. Scattered over the peritoneum and intestines were similar tubercles but becoming more sparse in the lower parts, as if they had been disseminated from the region of the diaphragm on both sides, for the same condition of tubercular growth was found on the left side also. There were, however, no tubercles whatever in the liver, lungs, kidneys or spleen. The tubercular deposit increased as the region of the suprarenal capsules was reached, and here, embedded in dense fibroid tissue, the capsules were found after much dissection. They were of normal size but their structure was entirely destroyed. For the most part they consisted of translucent fibrous tissue, in the midst of which were small nodules of caseous change, most of them firm and not softening, with here and there a few small calcareous nodules. The solar plexus and nerves could not be found, for they were embedded in fibrous tissue as the capsules were. The heart was small relatively to the body. The condition of the capsules presents no special interest further than that it is exactly that which is described as typical of the disease by Greenhow.[1] A more interesting fact is the existence of chronic tuberculosis in the immediate neighbourhood of the capsules and in its dissemination from thence over the peritoneum, there being no general infection of the body. The condition of the liver was peculiar and, as far as I know, rare, for the tuberculosis, though of some standing and well developed, was entirely confined to the capsule.

In respect of age the case deserves record, for in Dr. Greenhow's cases only one out of six males were under twenty years of age, and this patient was only seventeen. The duration of the case was also short,—the lad was in good health and at work eight months before his death. The bronzing was noticed one month later. Dr. Wilks puts the average duration of the disease at eighteen months.

The last point of interest was the rapid development under observation of black spots and patches of pigment.

November 1*st*, 1887.

P.S.—The capsules were examined for tubercle bacilli by Dr. Sidney Martin, but none were found.

6. *Tubercular disease of the suprarenal capsule unattended by bronzing of the skin.*

By William Collier, M.D.

The right suprarenal capsule exhibited was taken from the body of a female patient aged 36, who was recently admitted into the Radcliffe Infirmary. Her illness had commenced about nine months before with pain across the loins, occasional attacks of vomiting, feeling of weakness and exhaustion; these symptoms had continued and increased in severity. She stated that about six weeks before admission she had vomited a small quantity of blood. Her previous health, with the exception of an attack of rheumatic fever at the age of twenty-one, had been very good. Patient was a well-nourished, fair-complexioned woman, decidedly anæmic, with an extremely weak pulse. No disease of any of the organs could be discovered, but on palpation there was decided tenderness over the loins. While in hospital patient vomited occasionally, but did not complain particularly of pain in the stomach. She was extremely languid, lying in bed and taking no notice of anyone or anything. On more than one occasion a note was made of the extreme feebleness of the pulse. Temperature during first week rose to about 100° F. towards evening but afterwards fell to and continued normal. Patient was dieted carefully, and bismuth and iron given under the impression that she might have an ulcer of the stomach. One afternoon about two weeks after admission, patient, while sitting up, became suddenly faint and collapsed. Ether was injected and she rallied but died three hours later in a similar attack.

Autopsy.—Great excess of subcutaneous and peritoneal fat noted; mesenteric and lumbar glands were enlarged and some were the seat of caseous deposit. Both suprarenal capsules were enlarged, more particularly the right, in which the normal tissue had entirely disappeared and had given place to a cheesy, yellowish deposit, which had become in parts cretaceous. On section of the organ an irregular cavity was found containing thick creamy fluid. The only trace of the left suprarenal was a small sac enclosing cheesy material, which was densely adherent to and could not be separated

from the under surface of the pancreas. At the point at which the right suprarenal was adherent to the liver that organ was invaded to the depth of about an inch by a caseous deposit.

The symptoms in this case were very misleading, as the patient had not the slightest trace of bronzing or abnormal pigmentation in any part of the body. The anæmia, vomiting, and history of having on one occasion vomited a small quantity of blood, were suggestive of gastric ulcer. On the other hand, I entirely misinterpreted the extreme weakness of the radial pulses, hastily concluding that it was due to some abnormality of the radial arteries. Cases in which the disease has existed for so long a period without producing bronzing of the skin are decidedly rare, as, out of a series of 228 cases of Addison's collected by Dr. Greenhow, only eleven were free from pigmentation, and in each of these cases the patient's illness had been of short duration; in one only had it lasted eight months, in another four months.

March 20th, 1888.

IX. DISEASES, ETC., OF THE SKIN.

1. *On some examples of Arsenic-Keratosis of the skin and of Arsenic-Cancer.*

By Jonathan Hutchinson, F.R.S.

(With Plates XXI, XXII, and XXIII.)

In the following statement I have two separate propositions to maintain, one of which is, however, of great importance to the other. The first is that by the prolonged internal use of arsenic the nutrition of the skin may be seriously affected, and that, amongst other changes, warty or corn-like indurations may be produced. The second goes much further and asserts that if the drug be continued these "arsenic corns" may assume a tendency to grow downwards and pass into epithelial cancer.

It will be convenient to begin with to produce evidence as to the first of these assertions. A few years ago a gentleman of nearly sixty was sent to me by Dr. H. Weber on account of an eruption which had apparently followed the use of arsenic. He was the subject of malignant disease of the right tonsil and glands of neck, and in order to restrain its growth arsenic had been prescribed in large doses. For several months he had been taking regularly Fowler's solution in twelve-minim doses three times a day. Under its employment he had emaciated, had suffered much from stomach neuralgia, and his skin, which previously had been perfectly healthy, had become everywhere of a brown, earthy tint, and covered in parts by scaly patches like psoriasis. The scaly condition was most obvious on his elbows and backs of knuckles. The drawings now exhibited show the state of these parts very correctly. The palms of his hands had become dry, slightly horny, and covered by little pits

DESCRIPTION OF PLATE XXI.

In illustration of Mr. Hutchinson's paper on Arsenic-Keratosis and Arsenic-Cancer. (Page 352.)

The Plate shows the elbow of a gentleman whose hands are delineated in Plate XXII. It will be seen that the whole skin is of a dirty brown tint and that a large scaly patch has been developed near to the elbow. This patch was quite dry, but it was somewhat less circumscribed and showed more of redness about its edges than common psoriasis usually does. The patient had a perfectly healthy skin previous to the administration of arsenic.

DESCRIPTION OF PLATE XXII.

Illustrating Mr. Hutchinson's paper on Arsenic-Keratosis and Arsenic-Cancer. (Page 352.)

The back and front surfaces of the hands of Mr. D—, the patient whose elbow is shown in the preceding plate. It will be seen that across the knuckles of the hand a number of dry, thick, scaly crusts have formed. On the backs of the fingers there are numerous little pits where the epidermis is broken, while the skin generally is discoloured and harsh. The patches across the knuckles are exactly like psoriasis, excepting, perhaps, that there is more thickening beneath the crusts. They do not occur precisely in the parts where psoriasis is most common, that is, on the back of the hand, rather than the knuckles. The palmar surface of the hand shows the epidermis dry and cracked, and there are numerous little pits where it has been broken. The extremities of the fingers are especially affected and somewhat reddened. It has been difficult to represent the little corn-like patches of thickened epidermis which were present.

DESCRIPTION OF PLATE XXIII.

Illustrating Mr. Hutchinson's paper on Arsenic-Keratosis and Arsenic-Cancer. (Page 352.)

The palmar surfaces of the two hands of Dr. W—, who became the subject of arsenic-cancer. It will be seen that the palms are exactly like that of the hand shown in Plate XXII, but with the addition of a fungating growth in each. That in the right palm is of considerable size and thickness and is placed just above the wrist. That in the left is much smaller, having been of more recent development, and shows only a reddened excoriation between the index and middle fingers, beneath which there is a certain amount of thickening. In this case common psoriasis had been present in the first instance, and arsenic had been given in large doses over a long time for its cure.

where the epidermis had broken up. In some spots the epidermal layers were thickened into small hard corns (see Plates XXI and XXII).

The arsenic was at once discontinued, with the result that the stomach pains ceased and that the state of the skin was definitely ameliorated. The drug was afterwards resumed and the skin got worse. This patient, who was, as already stated, the subject of cancer before the arsenic was given, never developed any fresh cancer in connection with his arsenic corns. He died of his original disease a few months after I first saw him. The point which I wish to establish in connection with his case is that the state of skin was without question due to arsenic, and that it was exactly like that which, in the next case proved to be introductory to cancer.

The subject of the next case was a well-known member of the medical profession in Boston, U.S. His case has already been ably recorded by Dr. James C. White, of Harvard University, under the title of "Psoriasis—Verruca—Epithelioma: a Sequence." I have nothing of importance to add to Dr. White's narrative, excepting the suggestion that the verrucæ, or rather corns, which preceded the epithelioma, were themselves caused by the long-continued use of arsenic for the cure of the psoriasis. Briefly, the facts are these: Dr. W—, then aged 44, came over to England for advice in December, 1883, with his hands in the condition shown in plate (Plate XXIII). With the exception of the addition of new growths (cancer) they were the counterparts of the hands of Mr. D—, the subject of the preceding case. There was the same general dryness, the same tendency to form little pits in the hardened epidermis and to produce little dense corns.[1] Dr. W— had been the subject of psoriasis from boyhood, and had taken large quantities of arsenic. The psoriasis, which was of the ordinary form, did not originally affect his palms (a very rare event), and the state observed in them had only come on after long use of the drug. On this important point Dr. W—'s testimony was clear. The cancerous processes were yet more recent and had been present in the right palm about three years before I saw him. As Dr. White witnessed the whole

[1] Dr. White's description of these warts or corns is as follows: "Sharply defined, prominent, firm, and horny outgrowths, some of them resembling the unpigmented formations of keratosis senilis in the same localities, whilst others were more like callosities or some form of warts."

process of development, it is better that it should be described in his own words:

"Three years ago one of these outgrowths, situated upon the right palm near the wrist in one of the great longitudinal dividing furrows of the skin, became excoriated, either by abrasion or fissuring, which refused to heal by the simple measures employed, and terminated in a small ulcer. It remained in this condition many months, sometimes nearly filling up and protecting itself with a thin epithelial cover, sometimes breaking down again and remaining in an open indolent condition, but the destructive process gradually extended more deeply into the cutaneous tissues, and widened its borders. After a time the peripheral portions of the integument became indurated and thickened, forming a dense and elevated circumvallation about the central ulceration. During this long period the efforts to restore the part to a healthy condition were persistent and various; at first simply soothing applications combined with restraint to the movements of the part, afterwards more stimulating applications, then cauterizing agents, as chromic and concentrated nitric acids, and later the curette. Finally, as the ulcer enlarged and deepened, so that the integument involved in the destructive process and surrounding induration was one half inch in area. The whole growth, in August, 1883, was deeply scraped. At the same time another lesion, identical in history and character, but of much smaller size, which had more recently and gradually established itself upon one of the warty hypertrophies situated upon the palmar fold of skin between the fore and middle fingers of the left hand, was also thoroughly curetted."

Dr. W—, whilst in London, saw Sir James Paget and myself, and microscopic sections, which he brought with him, were carefully examined by us, and were also made the subject of a critical report by Dr. Creighton. The conclusion arrived at was that they were certainly epitheliomatous. He next went on to the Continent and obtained the opinions, amongst others, of Professors Billroth and Kaposi at Vienna. He afterwards returned to New York, where, in accordance with the recommendations given him, his right forearm was amputated. This was done by Dr. Hodges, and at the same time the affected fingers of the left hand were removed.

The sequel proved the malignancy of the disease, for he died

about sixteen months after the amputation with growths in the axillary lymphatics and internal organs.

I quote from Dr. White's paper the description by Dr. Fitz of the examination of the amputated parts.

"The palm of the right hand presented an elevated, rounded, ulcerating mass with dense everted edges and irregularly scalloped outline. The surface for the most part was smooth, reddish-gray, and translucent, but showed an irregular, deep, sinuous depression at the upper and outer fourth. It measured two and a quarter by three and a third inches, and the mass projected two thirds of an inch above the cutaneous surface.

"On section the superficial ulcer corresponded with a circumscribed new formation, one and a quarter inches in thickness, extending through the skin and subcutaneous fat tissues to the deep fascia, being intimately united to the tendinous sheaths, which were not perforated. The growth was continued into the substance of the unciform bone and into the abductor minimi digiti. The cut section was in general relatively homogeneous, grey, and translucent. Minute ecchymoses were present near its free edge, and occasional opaque lines extended upwards and outwards from the base. Pressure caused the escape of small, soft, opaque white plugs.

"The microscopic examination showed that the structure was composed of variously shaped, anastomosing bands of cells, resembling the deeper layers of the epidermis, and separated by a framework of fibrous tissue. The bands contained numerous onion-shaped bodies of laminated epidermoid cells, and extended irregularly in all directions.

"At the palmar base of the fore and middle finger of the left hand was a small superficial ulcer upon the surface of a dense rounded and flattened nodule, one by one and a half inches in length and breadth, and three fourths of an inch in depth. The cut section of the nodule showed a grey, slightly translucent new formation, traversed by occasional fibres. The growth extended into the subcutaneous fat tissue, and the tendons beneath were freely movable. The structure was like that of the growth on the right hand, though the fibrous portion was relatively more abundant, and in both the characteristic appearances of flat cell epidermoid cancer were presented."

The appended letters which give full details as to Dr. W—'s last illness and the autopsy were kindly sent to me by Professor

Bigelow.[1] In addition to the information which they give Prof. Bigelow states that for some days before death there was acute pain in the cervical vertebræ. This region was not examined at the

[1] Boston; April 26th, 1886.

My dear Professor Bigelow,—The principal facts in the case of Dr. J. H. W— are these, viz.:—That about a month after seeing Mr. Hutchinson, in London, his right arm was amputated in the middle of the forearm. At the same time the fore and middle fingers of the left hand, with parts of their respective metacarpal bones, were also removed. These wounds healed rapidly. The cicatrices and tissues in the vicinity of both operations remained perfectly healthy until the patient's death. Eventually a lymphatic gland, enlarged to the size of a nutmeg, appeared above the left elbow. This was freely excised. Under the microscope this new growth was shown to be an epithelioma. The operation was quickly recovered from. A few months after its performance a severe and sudden "pleuritic" attack, unaccompanied by decided physical signs, confined Dr. W. to his bed for a few days. Although of brief duration, this sickness was followed by a dry and hacking cough, which never disappeared. In the latter part of 1885 a neuralgia, about the neck and right shoulder, wore upon the patient's strength and health, which had previously been excellent, and to the pain and exhaustion incident to its unremitting severity, he gradually succumbed at the end of the year.

At the time of Dr. W.'s death no reappearance of the local disease was visible externally, nor was there tangible evidence of its existence anywhere, except in an enlarged gland in the left axilla. I believe you have a report of the post-mortem examination, describing the internal lesions which were discovered.

I am, very sincerely yours,

Professor Henry J. Bigelow. R. M. Hodges.

Notes of the autopsy by Dr. Fitz.—The post-mortem examination was made 6th January, 1886. Body emaciated. In the left axilla was a dense flattened nodule, near, but not adherent, to the skin, as large as a dried fig. Nothing abnormal on examination of the pericardium and heart. The anterior surface of the left lung was adherent to the thoracic wall by recent adhesions. In the posterior and lower portion of the left pleural cavity were about three pints of opaque, reddish-grey fluid. The pleura was thickened, opaque, besmeared with a soft yellow false membrane. In the posterior and upper part of the left lower lobe was a firm rounded mass as large as a small orange. On section a central cavity was found, representing the greater part of the volume of the mass, and filled with an offensive, opaque grey fluid. The wall of the cavity was about a half inch in thickness, dense, yellowish grey in colour; its free surface was irregularly nodulated and granular. A dense, yellowish-grey nodule, without central softening, was found in the lower part of the left lung. At the root of this lung, but within the lower lobe, the main bronchus was surrounded by a dense neoplasm, resembling those already described, and extending outwards a half inch or more. The included bronchus was irregularly narrowed, and its mucous membrane deeply injected and infiltrated with blood. Elsewhere in the

post-mortem. It is very probable that a growth was forming there.

Dr. W—'s psoriasis began at about twenty, and he soon afterwards commenced the use of arsenic. Corns began to form on his hand when he was thirty-four, they ulcerated when he was forty-two, and the first scraping was done at forty-four. He came to England in the same year. The amputation was eight months after the scraping, and he died eighteen months after the former.

That the palmar keratoses were in this case the seats of the cancerous development no one will, I think, hold doubtful who reads Dr. White's description of the early stage of the latter, and who examines the portraits of the two hands. It will be seen in the portraits that the conditions in each are precisely similar, and thus we had a double illustration of the facts. That the keratoses were in their turn due to arsenic no one will, I think, doubt who has given attention to the preceding case.

I have next to mention three other cases from my own experience in which cancerous processes followed the use of arsenic. Two of these are of the more value because they occurred long before I had

lungs nothing abnormal was found with the exception of œdema. The bronchial glands were not abnormal in appearance.

In one of the ribs of the left thorax, near the spine, was a central nodule of new formation, as large as a walnut, apparently myologenous, which had caused an expansion of the rib, with an absorption of the lime salts in the cortex.

Nothing abnormal found on examination of the spleen. The left kidney, not particularly enlarged, contained numerous round nodules, dense, opaque, grey, varying in size from that of a pea to that of a marble. The right kidney increased in size, at least one third, presented similar changes, the nodules being more numerous and larger. A nodule of new formation, as large as a marble, was found in the perinephritic fat tissue in the vicinity of the right renal artery, and some of the lumbar lymphatic glands were moderately enlarged and indurated. The right suprarenal capsule was tranformed into a dense yellowish-grey mass, as large as a prune. The left capsule apparently normal. The upper surface of the right lobe of the liver showed a grey nodule, as large as a dried pea. There was a moderate degree of fatty infiltration of the liver.

The microscopic examination of the nodules in the lung and kidney showed an alveolar structure. The alveoli were often small, the stroma abundant and dense. The contained cells were epithelioid and small.

Diagnosis.—Cancer of the left axilla, of the left lung, of a left rib. Cancer of the kidneys, right suprarenal capsule and perinephritic fat tissue, lumbar lymphatic glands and liver. Acute pleurisy; œdema of the lungs.

(Signed) R. H. Fitz, M.D.

formed any opinion as to the action of arsenic in this direction. I have taken them out of old note-books, where they had been recorded without any special theory in view.

In the first of these the palms were affected by corns, but the cancer occurred on the scrotum. A clerk named Edward B—, aged 34, was at the Hospital for Skin Diseases in August, 1871. He had been long under Mr. Hunt's treatment by arsenic for psoriasis. There was cancer in his family. The palms of his hands were speckled with numerous whitish spots, which were slightly raised, "just like so many soft corns," "no warts." The corns were specially marked in the palms and where the fingers rubbed together. The soles of the feet were in a similar condition. He had noticed this condition for eighteen months, and it began after he had been some years under Mr. Hunt's treatment.

At the Hospital for Skin Diseases we continued arsenic, not then knowing any better.

In February, 1872, he had a hard-edged sore on the scrotum, which had been preceded by a wart or corn. In June this sore was recognised to be epithelial cancer, and early in July he was taken into the London Hospital, and had it excised. At this date the corns in the palms had to some extent "peeled off."

In April, 1873, there was no return and no enlarged glands.

I am sorry that I have not been able to obtain more recent information as to this patient.

The following notes concern a patient whom I never saw, but about whom I was consulted by letter by Dr. Clifford Allbutt, of Leeds. The lady died not long after the notes were written.

Miss H—, when aged 5, came under Dr. Clifford Allbutt's care for pemphigus. It was a very bad case. She took arsenic, and it stopped the eruption. The latter, however, relapsed, and she got into the way of taking arsenic herself. The inner aspects of her hands and fingers became "very rough." She married before twenty, and had a child born.

When twenty-five she came again to Dr. Allbutt with a large, open ulcer behind the crest of the ilium on left side. She said that there had been a roughness on the spot, and that she believed the sore had been caused by her clothes hitching. There was a secondary glandular swelling in the groin. Subsequently a lump formed in the skin of the inner aspect of the thigh, "hard, yellowish reddish and purple mottled, but not pigmented." At this stage the gland

mass had ulcerated, and it, as well as the original, were described as like a tomato in colour and shape, having lumpy, everted edges.

No opportunity for microscopic examination ever occurred, but that the patient died of malignant disease, sarcomatous or epithelial, there could be no doubt. There was equally little that her skin had been made rough by the long-continued use of arsenic.

Another example of cancer following keratosis came under my notice in the person of gentleman, aged 55, from Edinburgh. In him the growth had occurred on the sole of the foot, and the appearances were closely similar to those exhibited in the portrait of the Boston case. (A portrait of sole of foot was shown at the meeting.) Anyone looking at the portrait might be inclined to suggest "perforating ulcer," for the ulcer is in exactly the position in which that ulcer usually occurs, and it has thickened edges which are not dissimilar in general appearance. It was, however, most certainly not of that nature, for the ulceration did not pass deeply and there was definite new growth around it.

The history which I received with this patient was that he had for many years been the subject of psoriasis and had taken much arsenic. "For many years he has been troubled with a tendency to formation of corns on hands and feet and general hypertrophy of cuticle. The skin becomes dry and harsh. He has lost to a great extent the power of feeling in the right hand, so that he does not know when he is grasping a pen, and has to write with his left hand." In December, 1886, the following note as to the state of his foot was made: "The skin of the sole of the foot is dry and rough, and over the ball of the great toe there are two ulcers the size of three-penny bits, the skin around which is much thickened." . . . "The patient states that these ulcers are in the place where for several months he had two hard corns which he used to have pared." That the ulcers which Mr. — suffered from were of no ordinary kind may be assumed from the variety of measures of treatment which they had resisted. In addition to numerous local applications the foot had been kept at complete rest in a plaster-of-Paris case for some months and walking had been wholly forbidden, yet no tendency to heal was shown. It was on account of this obduracy that he was finally sent to London.

This gentleman was under the care of my friend, Prof. Chiene,

in Edinburgh, and he returned to him and had the affected part freely excised and treated with chloride of zinc. Microscopic examination of the portions removed did not reveal any elements which were conclusive as to epithelial cancer, but to this negative result I may freely confess that I do not attach much importance as regards diagnosis. After the scraping the sore healed, and it remains, I believe, sound up to the present time (about a year).

The microscopic examination was very carefully made by Dr. George Mackay. I extract the following sentence from his report, it being the one bearing most nearly on the question: "The sections show intrusion of epithelium at the margin but without rupture of boundary zone between epithelium and connective tissue. This may be nothing more than the natural growth of the epithelium and papillæ recently pointed out as tending to occur as life advances (Dr. Woodhead)."

On December 4th, 1887, Mr. Chiene wrote me: "I saw Mr. C— to-day; there is a corny cicatrix at seat of operation, but nothing more; no pain. The corns on that foot are better but the corns on the sole of the opposite foot and on the hands remain as they were—no improvement. He still suffers from psoriasis and has twice taken since the operation a little arsenic."

In Dr. White's paper, to which I have already been so largely indebted, another case is recorded from his own observation of an almost precisely similar nature. A gentleman, aged 52, had suffered from psoriasis all his life and had taken much arsenic. Ten years before coming under Dr. White's care he had noticed that his hands were beginning to assume a thickened warty condition. Some years later an ulcer formed on the front of the wrist which resisted all treatment. Caustics and scraping were freely employed, but in spite of them it passed into a condition which Dr. White describes as exactly like that which is shown in Plate XXIII. Dr. Hodges was also struck with the remarkable similarity between the two cases. A very free excision of the diseased parts was made. Some months later, soon after a second operation for removal of sloughing tendons, the patient sank from acute cellulitis of the arm. This patient had also a small growth near the anus and it was also excised. The most important paragraph in Prof. Fitz's histological report on the parts removed is the following: "The specimen was characterised by the presence of large masses of epithelioid cells of irregular shape and size, separated by narrow

bands of fibrous tissue and extending deeply downwards into the subcutaneous fat tissue."

Dr. White's diligent research has been rewarded by yet another case, which is recorded in 'Gazette des Hôpitaux,' 1878, p. 750. In this Dr. Cartaz reported to the Anatomical Society the case of a healthy man, aged 40, who had for twenty-three years suffered from psoriasis, which had, at length, among other regions, invaded the palms and soles. "In consequence of scratching, the scales from one of these processes, situated upon the palmar surface of the second phalanx of the ring finger of the right hand, were removed, and there remained a little ulceration which gradually extended to the size of 4 centimetres. The callous borders of the deep-seated granulations, bleeding at the slightest touch, established the diagnosis of *cancroide*. Amputation at the metacarpo-phalangeal joint was performed and there was no return of the disease." The narration of this case is defective in that we have no statement of the intermediate formation of a corn, and no information as to whether or not arsenic had been used. The fact that the man had suffered from psoriasis more than twenty years makes it very probable that he had taken it.

In the preceding statements six cases have been adduced in which cancer occurred in persons who had for very long periods suffered from disease of the skin. In five of these arsenic was known to have been very freely used over very long periods, and in the sixth there is no evidence to the contrary. In five of the cases the skin disease was psoriasis, but in the fifth it was pemphigus. In one case the scrotum was the part on which the cancerous ulcer formed and in one the side of the trunk, whilst in all the others it occurred on the hand or foot (sole once, palm once, and finger once). In all cases a condition of local keratosis preceded the cancer. One of the cases, that of Dr. W—, should almost rank as a double fact, for the keratosis and cancer occurred sympathetically in the two hands. Three of the patients are dead (two of secondary malignant growths), and respecting two others nothing is known as to later history.

The question to be considered is as to whether there be any real connection between the skin disease and the cancer following it, and if so what? The reply which I have suggested is that the bond of connection is to be found in the influence of the drug given for the cure of the skin disease. It seems highly improbable that psoriasis, however protracted, should in any number of cases result in cancer.

We have no clinical experiences to support such a conclusion. The parts affected in a majority of these cases are, besides, precisely those which psoriasis does not attack. It is exceedingly rare for common psoriasis to show itself on the palmar surfaces. Its favourite sites are the extensor surfaces, and when the hand is involved it is usually its back. Yet in most of these cases the palmar surfaces were affected, and keratoses, which are unknown as a part of common psoriasis, occurred late in the disease as a precursor of the cancerous process. In this difficulty the first case which I adduced comes to our aid with evidence which is invaluable. In it changes in the nutrition of the skin, and especially in that of the palms of the hands, were induced by the use of arsenic, and they were precisely like those which in the other cases preceded cancer. Nor does this case by any means stand alone as proof that arsenic can alter the state of the skin. Many others have been recorded in which the integument has become dry, harsh, and muddy during its use, and some in which a great increase of pigment was the result. It may be admitted that the formation of keratoses under its influence is a more novel observation, but it is only going a short step farther. It would appear that the influence of arsenic when used to saturation through long periods is to lower the nutrition of the skin and render it less able to bear ordinary kinds of irritation. Thus we find the palms and soles forming corns, from local pressure presumably, in a most unusual manner. The adjacent surfaces of the fingers where touching each other also form corns, and in one of the cases which I have recorded the part affected had been pressed upon by the dress. Not only does arsenic depress the nutrition of the skin, it may induce local neuritis. That it causes the outbreak of herpes is now, I think, generally admitted, and in one of the cases just mentioned the fact was recorded that the patient had a partial paralysis of one hand. I believe that I have seen more than one case in which arsenic has caused the formation of opacities in the vitreous humour.

In conclusion, a word or two may perhaps be permitted as to the clinical bearing of the facts adduced. Dr. White has very properly insisted that "the lesson to be drawn from their study is that the transformation of patches of psoriasis into verrucous hypertrophy must be regarded as an ominous occurrence, and that the softening or other change of such horny growths demand thorough excision without delay." To this I would venture to add that it is desirable

to take precautions still earlier, and that in all cases in which during the use of arsenic for any skin disease the palms and soles become rough and corneous it is an indication that the use of that drug should be laid aside. If the case when first seen is more advanced and cancer be already threatened, the propriety of amputation in preference to excision must be carefully entertained, for the form of cancer would appear to be one which is promptly infective to the glands and other parts. *December 6th*, 1887.

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2. *Experimental observations on the pathology of lupus, with notes on three cases of lupus or tuberculosis of the skin of the hand.*

By Frederic S. Eve.

[With Plate XVIII, figs. 3 and 4.]

The pathology of lupus has in recent years attracted much attention, and the advances made are familiar to most of us. The opinions of Continental authorities on the subject were fully exposed in the discussion which took place at the International Medical Congress in Copenhagen in 1884, when M. Leloir communicated a paper on the subject, in which he contended that lupus was a local form of tuberculosis of the skin. This proposition was combated by Kaposi and others. More recently Schwimmer, in a paper read before the German Congress of Surgeons, has followed the same line of argument as Kaposi. The latest contribution to the subject, by our greatest authority, Mr. Hutchinson (Harveian Lectures, 1887), is little more favorable to the tubercular theory of lupus. Mr. Hutchinson apparently considers the disease as a non-specific inflammation having locally infective characters. He says in effect that whilst some of the subjects of the disease have the strumous or tubercular constitution, a large number enjoy otherwise sound health.

Most of the arguments adduced on either side may be briefly stated as follows:

The view that lupus is a local tuberculosis of the skin is sup-

ported by these considerations: First, the identity of the histological structure of lupus and tubercle. Essentially lupus consists of granulation tissue; and, as Friedlander has demonstrated, giant-cells are sometimes present. Secondly, the existence of Koch's tubercle bacilli in lupus. Koch has stated that he cultivated lupus bacilli for sixteen generations without observing any difference between them and tubercle bacilli. Thirdly, positive results of inoculation experiments on animals. Guinea-pigs inoculated beneath the skin or in the peritoneum with lupus become affected with general tuberculosis. Fourthly, it is stated that transition forms exist between the varieties of lupus and of tuberculosis of the skin. Fifthly, tubercular manifestations are sometimes coincident with lupus, and it has been estimated that in ten cases of lupus one becomes affected with phthisis. Sixthly, that the disease has a tendency to spread, is locally contagious, and frequently recurs after destruction.

In answer to these assertions it has been urged by Kaposi, Schwimmer, and others: First, that giant-cells are found in simple inflammations, and are not characteristic of tubercle. Secondly, that the bacilli are exceedingly scanty in lupus, whereas they are very abundant in tuberculosis of the skin, and that other well-known pathogenic bacilli possess the same staining reaction, among them being the smegma bacillus of Alvarez and Tavel. But I may add that the smegma bacillus only possesses this reaction when grown in its natural habitat and surrounded by fat. Thirdly, that tuberculosis affects primarily the mucous membrane, and is usually a secondary manifestation in the skin, the reverse holding good in the case of lupus. Fourthly, lupus in man is rarely followed by general tuberculosis or affection of the internal organs. Fifthly, the different effect of treatment of the two diseases, lupus being often cured by scraping and caustics, whereas tuberculosis is not favorably influenced.

Schwimmer also remarks that while general tuberculosis has been developed by the inoculation of lupus in guinea-pigs, a local disease resembling lupus has never been produced. It has been stated by Leloir and others that rabbits are not affected by lupus, while they take tuberculosis readily, and I have shown that visceral tuberculosis may be communicated to rabbits by inoculation of the local form of tuberculosis known as strumous gland disease.

Although, as may be seen, much has been done to elucidate the pathology of this malady, some links in the chain still remain incomplete. I have brought forward the subject this evening with the view of showing that true lupus, as a local disease, may be produced artificially in animals.

On May 7th, 1886, I obtained the following positive results:—A large rabbit was inoculated beneath the skin of the ear with tissue from an undoubted case of lupus vulgaris of the nose, in which there was a good deal of papillary overgrowth. On May 20th the seat of inoculation was marked by a scab, beneath which was some caseous pus. Subsequently a large ulcer covered by a thick scab formed, and soon after an ulcer possessing precisely similar characters broke out at the root of the ear at the distance of one inch from the spot first inoculated. The ear was much swollen, œdematous, hot, and all the hair was lost. In time the scab separated, and healing took place, leaving thickened nodules beneath the skin on the surface of the ear corresponding to the position of the ulcers. The animal was killed on December 16th, 1886, and no trace of visceral tuberculosis could be detected. The lymphatic glands of the neck on the same side as the ear inoculated were healthy. (The ear was exhibited.) I did not obtain the next positive result with primary inoculation of lupus tissue until some time afterwards, and therefore remained doubtful whether that obtained in the first case was not accidental. On November 28th, 1887, I inoculated a large strong rabbit in the same manner in the ear. On December 3rd the ear was much swollen and œdematous. On the 8th the following note was made:—"The ear is still much swollen. At the seat of inoculation is a dry patch, over which the skin is superficially destroyed. Above this, but not continuous with it, is a round dryish ulcer, and on one side of this, again, is a blister of considerable size. The left ear was inoculated from one of the ulcers on the right."

13th.—The larger ulcer at the seat of inoculation has extended downwards, and is of a dry character. The smaller ulcer above is covered with a dry scab. A superficial ulcerating process, without distinct necrosis, has also appeared on the posterior margin of the ear, which has led to slight destruction. I removed the scabs, and Mr. Lewin made the accompanying drawing (Plate XVIII, fig. 4), which faithfully represents the condition then observed. Near the base of the ear is a long oval ulcer with a

dryish, dusky-red surface, and somewhat undermined margins, to which a few scabs are adherent. This was the site of the inoculation. Some distance above and entirely disconnected with it, is a smaller ulcer having the same characters. It may be seen that the posterior margin of the ear is superficially eroded. On the left ear is a rounded nodule filled with caseous pus produced by inoculation with a scraping from the ulcer on the right ear. On the 30th the ulcerations on the right ear were cicatrised, leaving very considerable thickening (the ear was exhibited). On January 5th, 1888, no ulcer had formed at the site of inoculation on the left ear, but a superficial ulceration precisely like that observed on the right had commenced on its posterior margin. Between it and the caseous abscess were white nodules and white ramifying thickenings or cords apparently marking an extension of disease along the lymphatics of the ear. They reminded one of the tuberculous lymphangitis observed by Leloir in connection with a case of lupus of the left hand (see 'Annal. de Dermat. et Syph.,' 1886, vol. vii).

It can hardly be denied that the disease produced in the ears of these two rabbits was true lupus. The inoculation was followed by spreading ulceration covered by a dry scab. The disease was not only locally contagious but the contagion was transmitted to more or less distant parts, for in each case ulcers formed apart from the site of primary inoculation. A considerable destruction of skin took place and a depressed scar resulted. In the second case nodules and cords were observed in the neighbourhood of the ulcer, which might perhaps be considered analogous to the gelatinous deposits around lupus of the face in the human subject. On December 7th, 1887, a young rabbit was similarly inoculated. On the 13th the skin at the site of inoculation appeared to be dried up, and there was a corresponding swelling on the inner side of the ear. Subsequently a dry ulcer formed which began to heal on the 30th, and cicatrisation was completed on January 10th, 1888.

I also inoculated two rabbits with the organs of a guinea-pig to which tuberculosis had been communicated by placing pieces of lupus in the abdominal cavity. There were miliary nodules in the liver. The spleen was much enlarged and filled with nodules of caseous matter, and both testicles were affected with well-marked tuberculosis. One rabbit, inoculated in the ear, developed a perfectly dry, superficial ulcer, which extended with an undermined margin.

No suppuration or scabbing took place, and the rest of the ear remained unaffected.

The other rabit was inoculated in the anterior chamber of the eye and in the ear. A caseous nodule formed on the ear, but no ulceration occurred. The mass in the eye did not grow, and produced no inflammation. This animal was killed after the lapse of six months, and was found to be perfectly free from visceral tuberculosis; the other is still living. It would therefore appear as if the virulence of the disease was not increased by passing it through the guinea-pig, as is the case with most local tuberculoses, and the results obtained resembled rather those sometimes following inoculation of strumous glands beneath the skin of the ear of rabbits rather than those of my previous experiments with lupus. Briefly to sum up the results of these experiments it may fairly be contended that lupus, as such, has been produced in rabbits, and the missing link in the chain of evidence referred to by Schwimmer has been supplied. This appears of much greater importance as evidence of the close relation of tubercle and lupus than is manifest at first sight.

In the human subject lupus is modified by the age and constitution of the patient, and also according to the vital energy and activity of the circulation in the part affected. But the different degrees of susceptibility of various animals to the tubercular virus enable us to obtain still wider diversities than are usually observed in man. Thus, in guinea-pigs, which possess in the highest degree a tuberculous constitution, we have general tuberculosis almost invariably produced by inoculation with lupus. (In one of my experiments there was also a dry ulceration in the groin which was the seat of inoculation. I readily demonstrated, by means of Neelsen's method, tubercle bacilli in small numbers in the tuberculous lesions of the viscera of this guinea-pig.) In rabbits, which possess in a lesser degree the tuberculous constitution, we have a destructive and locally contagious ulceration without constitutional affection. These facts, together with the knowledge that a certain, although perhaps small, proportion of lupus patients do become affected with phthisis and local tuberculosis, render it difficult to escape from the conclusion that lupus is a modified tuberculosis. The circumstance that by the direct inoculation of lupus in the ear of rabbits in some cases an extensively destructive ulceration was produced, whereas by the

inoculation of lupus passed once through the guinea-pig only a slight superficial ulceration or cold abscess resulted, calls for some comment. It suggests to me that the ulcerative character of some forms of lupus may depend on a mixed infection with perhaps tubercle bacilli and some form of micrococcus favouring suppuration and ulceration.

It has been stated that micrococci are not found in lupus. I was, however, able to stain large masses of micrococci in the specimen of lupus with which the experiment first mentioned was performed. I regret that my present opportunities rendered it impossible for me to follow out the experiments bacteriologically. Since, however, Koch has demonstrated and cultivated bacilli, identical with those of tubercle, in lupus there is little encouragement for other observers to prosecute inquiry in that direction.

I now turn to some clinical aspects of the subject pointing to a close relationship between lupus and tuberculosis of the skin. Doutrelepont has recently placed on record two cases ('Arch. f. Dermat.,' Heft i, 1888, p. 98) in one of which lupus and tuberculosis of the skin existed in the same individual; in the other, lupus, scrofuloderma, and tuberculosis. Within the last month I was much impressed by two patients, both boys, who came under my care. In one angular curvature of the spine, lupus vulgaris of the face and phlyctenular ophthalmia coexisted; the other also had angular curvature and a lupus or tuberculous nodule on the back of his left hand. I excised the nodule. This and the two subjoined examples of tuberculous lesions of the hand seem in many respects to stand midway between lupus and tuberculosis, and may merit a brief description.

Case 1. *Angular curvature and lupus? on dorsum of hand.*—The nodule formed a distinct, raised, firm, plaque, the size of a half-crown piece. Its edges shelved abruptly. Its surface was dark red, slightly rough, with evident thickening of epithelium, but not ulcerated. It had remained unchanged for some three or four months. The microscope showed merely dense infiltration of the papillary layer and derma with leucocytes (granulation tissue) which extended down only as low as the sudoriparous glands. The cuticle was thickened, and the interpapillary processes of epithelium were prolonged downwards. There were no giant-cells, no reticulum, no caseous degeneration. Several sections were examined for bacilli but none were found.

Case 2. *Lupus? of palm of hand.*—This interesting specimen was brought by Mr. Bryant to the College of Surgeons. It comprised a nodule which he had removed from the hypothenar eminence of the palm of the right hand of a man aged 26. Four years previously a tumour appeared at this situation, and was excised eighteen months before the present operation. This growth was examined elsewhere, and stated to be a fibroma, and recurrent growth formed, and was believed, from its appearance and the general circumstances of the case, to be a melanotic sarcoma. The skin covering it was of a faint black tint, raised, and on section a firm material was found to occupy the derma and subcutaneous tissue. Microscopic sections exhibited a nearly uniform mass of reticulated tissue covered by a layer of slightly thickened epithelium. In parts the remains of giant-cells, which had not become completely incorporated with the reticulum, and areas where the reticulum was composed of the broad homogeneous bands so characteristic of tubercle, were observed. But for the most part it was indistinctly fibrillar, forming close meshes, enclosing small nuclei (reticular tubercle).

Case 3. *Papillary tuberculous formation on dorsum of hand.*—This specimen was presented to the College Museum in 1875 by the late Mr. Gay. At that time it was examined by one of my predecessors, and on account of the overgrowth of epithelium the disease was thought to be epithelioma. I subsequently described it in the catalogue (No. 4078) as follows:—"A hand, on the back of which a patch of skin near the bases of the second and third fingers is thickened and covered with scaly crusts and papillæ. There is no thickening around the patch, but the skin was livid in the recent state. The third finger is represented by a stump, and at its extremity are several separate masses of low papillæ." No history is obtainable; but amputation of the ring finger was probably performed for the same disease as that on the back of the hand. Section of the growth, recently made, showed under the microscope many tubercular areas, containing one or several giant-cells (see Plate XVIII, fig. 3). These occupied the papillary layer and corium. The cuticle was much thickened, forming several layers of cornified epithelium. The interpapillary processes of epithelium were extensively prolonged downwards. The rete Malpighii was infiltrated with groups of leucocytes; and at one point a giant-cell, surrounded by its zone of leucocytes, was completely enclosed

in epithelium. Mr. Shattock has described a similar case in the last number of the 'Transactions' (vol. xxxviii, p. 410).

Remarks.—The first case may probably be considered as a tuberculosis of the skin secondary to tuberculosis of the spine, although its appearance, course, and microscopic characters are suggestive of lupus. The second case agrees in its characters rather with lupus than with tuberculosis of the skin. The third case should perhaps be considered as tuberculosis, although its naked-eye appearances are very like lupus. The same condition has apparently been named by different writers lupus verrucosus and tuberculosis verrucosa cutis. *March 20th*, 1888.

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3. *Furrows on the nails after sea-sickness.*

By Samuel Wilks, M.D., F.R.S.

[With Plate XIX, fig. 3.]

Mr. L—, aged 50, a healthy, well-grown, robust man. He sailed for America on August 28th, 1887, and was very sea-sick for three days. He then quite recovered, and remained well until his return voyage on October 18th, when he was again ill for three days. He soon rallied as before, and has been in good health ever since. Some weeks after his return home he discovered two horizontal notches on his nails, and on making inquiries as to the rate of the growth of the nail found that these corresponded in date to the times of the sea-sickness. All the nails on both hands were affected. The photograph which he had taken on Feb. 8th is not very defined, but on examination of the living nail, and taking Mr. L—'s own impression of the facts, the lower mark corresponding to the illness of October 18th is about the middle of the nail, and quite above the lunula. The next groove, which occurred on August 28th, is about a quarter of the nail's length above, leaving the remaining quarter between this and the edge. According to this calculation, which is Mr. L—'s own, the nail would take seven months for its complete growth.

In my earlier observations on this subject I said that a groove in the middle of the nail generally indicates the occurrence of an illness three months before; that is allowing the growth of the nails two full lengths a year. Probably it is not quite so rapid as this.

March 20th, 1888.

X. SPECIAL COMMUNICATIONS.

1. *An investigation into the so-called Hendon cow disease in its relation to scarlet fever in man.*[1]

By Edgar Crookshank, M.B.

[With Plates XXIV—XXXIII.]

Before entering into the details of my investigation on this subject, I will lay before you a concise account[2] of *a vesicular disease of the teats of cows* which was discovered at a dairy farm in Hendon, and investigated jointly by Mr. Power, Dr. Cameron, and Dr. Klein.

The so-called Hendon Cow Disease and its alleged connection with an outbreak of Scarlatina in London.

On December 18th, 1885, Mr. Wynter Blyth reported to the Local Government Board a sudden and extensive outbreak of scarlatina, which was associated with the distribution of milk by a particular retailer in South Marylebone. The retailer procured his milk from two farms, and there can be little doubt that the milk responsible for the outbreak was derived from a certain dairy farm in Hendon. Mr. Blyth and the Medical Officer of Health for Hendon, Dr. Cameron, were unable to obtain any clue as to the

[1] Reprinted by permission from Reports to the Agricultural Department of the Privy Council.

[2] The account was published in Reports xv and xvi of the Local Government Board, in the 'Transactions of the Epidemiological Society,' New Series, vol. v, 'Proceedings of the Royal Society,' vol. xlii, and in 'Nature,' No. 919, vol. xxxvi.

source of infection, and Mr. Power was deputed by the Local Government Board to make an inquiry into the matter.

Mr. Power, having come to the conclusion that the milk had not become infected from any human source in any commonly accepted way, such as through unwholesome conditions of water or of drainage, *or through careless handling of milk or milk utensils by persons carrying scarlatina infection*, proceeded to investigate the condition of the cows.

A careful examination was made with a view to detecting any disease among the cows, and it was discovered that there were several on the farm which were suffering from a disease characterised by the appearance of *vesicles* and *crusts* on the teats and on the udder between or near the teats.

At this stage Dr. Klein became associated with Mr. Power in the inquiry, and we are told that their belief in the existence of a constitutional disease among the cows on the farm capable of producing scarlatina among human consumers of the cow's milk became unreserved.

Dr. Klein took away with him samples of milk, contents of *vesicles*, and discharges from ulcers, and afterwards two cows were purchased and conveyed to the Brown Institution. At this stage of the inquiry a cowman pointed out to Dr. Klein that he had seen the disease on a certain cow, which was one of a batch that had been imported on November 15th from Derbyshire.

Dr. Cameron has given a detailed description of the clinical history of this disease. He expressed his belief that it was a specific disease *capable of being communicated to healthy cows by direct inoculation of the teats with virus conveyed by the milker from a diseased animal.*

The condition of the teats is described as follows: The teats became enlarged, swollen to nearly twice their natural size, and œdematous. On fingering them there was no feeling of induration. Vesicles appeared on the swollen teats and upon the udder between or near the teats. These varied in number from two to four on a teat and in size from a pea to a horse bean. The vesicle contained a clear fluid. The vesicle, Dr. Cameron adds, was not preceded by a hardened papule, but was in the first instance a vesicle or bulla. The vesicles were rubbed and broken in milking and left raw sores, sometimes red, in other cases pale in colour, with raised, ulcerated edges. Sometimes a few accessory vesicles formed around the

margin of these ulcerated sores. After the rupture of the vesicle a brown scab formed, which might remain attached for five or six weeks, or fall off in ten days or a fortnight, a smaller one forming afterwards. A thin, watery fluid exuded from under the scab and the sore ultimately healed.

Dr. Cameron examined the teats of several cows five or six weeks after they were attacked. The scabs then varied in size from a shilling to a florin; they were about one eighth of an inch thick in the centre, thinning off towards the edges.

Some of the cows were also suffering from an eruption on the rump and hind quarter, consisting of patches of eczematous-looking crusts. When a crust was picked off the hair came off with it, exposing a raw, moist sore, the crusts and sores looking exactly like eczematous scabs and sores.[1]

In addition to his own observations, Dr. Cameron obtained information from the farmers, and others familiar with cows, who thought they recognised in the disease at the farm one stage of a disease which they were able to describe as a whole. Dr. Cameron thus gives an account of what he and his informants together would regard as a connected clinical history of the disease.

Dr. Cameron did not see the earlier symptoms, and these were of necessity, therefore, learnt from other persons. The account, therefore, of these symptoms was to be held liable to future correction or modification.

In the course of the inquiry, Dr. Cameron adds that it was strongly asserted by several people, who examined the cows, that they were suffering from cow-pox.[2] The disease was certainly not

[1] This concomitant condition in some of the animals corresponds in description with eczema, the result of ringworm, and other skin affections very common in cows.—[E. M. C.]

[2] Dr. Cameron, however, dismissed the diagnosis of cow-pox on the ground that no papule had been observed or subsequent formation of pustule, areola, or pitting, and because the vesicles were not umbilicated. I would take this opportunity of stating that the history of the Bordeaux cow-pox, which it is my intention to publish, illustrates that the reasons given for dismissing the diagnosis of cow-pox at Hendon were inadequate; a comparison having been made between the characters of the eruption of vaccinia as it appears on an infant's arm, instead of the eruption of the natural or so-called spontaneous disease on the teats of the cow. And I must add that the other vesicular diseases of the teats known to veterinary surgeons, not only were not eliminated by Dr. Cameron in the diagnosis of the disease at Hendon, but did not apparently claim any attention (see my papers in 'Brit. Med. Journ.,' July 7th and 14th, 1888).

a new one, as it had been known to some farmers and cow-keepers as a catching malady under the designation of sore teats, blistered teats, and the like.

Dr. Cameron states that the disease *was capable of being communicated to milkers by inoculation with virus from the vesicles on the teats*, while adding that seemingly it was communicable, *though perhaps in another form*, through the medium of the milk. An example of inoculation of the hands of a milker is thus briefly stated. "A trusty informant received the virus into a recent scratch on the forefinger while milking a diseased cow. General weakness, malaise, and loss of appetite resulted, and after about four or five days a vesicle or small blister appeared on the finger. This broke, and several others formed on the back of the hand. The whole hand and fingers became swollen and inflamed, the inflammation extending in broad lines as far as the elbow. The general disturbance lasted a fortnight."

I will now follow with the account given by Dr. Klein. He states that on the teats and udders of two cows which he investigated there were several flat irregular ulcers, varying in diameter from one quarter to three quarters of an inch. Some ulcers were more or less circular, others extended in a longitudinal direction on the teat. The ulcers were covered with a brownish or reddish-brown scab. The animals looked thin but not strikingly so. In feeding capacity, milking power, and body temperature there was nothing abnormal.

To study the transmissibility of the disease Dr. Klein inoculated four calves in the corium of the groin and the inside of the ear with scrapings from the ulcers after removal of the crusts. In calf No. 3, which may be taken as an example of the result obtained, there was vesiculation at the margin of the spot inoculated, and in the centre the commencement of the formation of a crust. On the seventh day each sore on the ear had enlarged to about half an inch in breadth, and was covered in its whole extent by a brownish crust. On the eighteenth day they had all healed up and become converted into flat scars.

To search for micro-organisms, Dr. Klein removed the crust from an ulcer on the teat, scraped off the most superficial layer, squeezed the ulcer, and made cover-glass preparations with the exuded lymph. Tubes of nutrient gelatine and nutrient agar-agar were also inoculated. Dr. Klein succeeded in isolating a micro-

coccus which, after three to six days' cultivation at 20° C., formed at the line of inoculation small points or granules, whitish in colour, and tolerably closely placed. In the depth of gelatine the channel of inoculation was visible as a whitish streak made up of smaller and larger droplets. The gelatine was not liquefied. Cultivated in milk and incubated for two days at 35° C. the milk was turned solid. Microscopic examination of a culture showed that there were micrococci, diplococci, and shorter and longer chains. In both media, after two or more weeks' incubation, very long chains were found. The chains showed branchings, and here and there in a chain the presence of a larger element amongst the smaller ones.

Dr. Klein inoculated two calves in the groin with the cultivated micro-organism. One calf died in twenty-seven days. No ulcers resulted, as after inoculation with the scraping of the sores, but at the necropsy there were found peritonitis, hæmorrhagic spots on the omentum, congested liver and kidneys and lungs, petechiæ under the pleura and pericarditis. In the second calf, on the nostrils and lips and on the hard palate and gums numerous irregularly outlined patches were noticed. They were discoloured, brownish, with a very slightly raised margin and a paler centre. This animal was killed, and at the necropsy there were congested lungs, kidneys, and hæmorrhagic patches on the spleen.

In these cases the *post-mortem* appearances and anatomical features recalled to Dr. Klein the lesions of scarlatina. In the kidney, for example, there were congestion of the cortex, hæmorrhages and glomerulo-nephritis, and granular or opaque swelling of the epithelial cells and infiltration with round-cells. From the blood of the heart the micrococcus, which had been used in the inoculation, was recovered. In view of this evidence Dr. Klein concluded that the streptococcus was identical with the virus of the cow disease, and that it produced a disease very closely resembling that of scarlatina in man.

Two of the cows selected from the Hendon farm were killed, and the following *post-mortem* appearances discovered:

Cow No. 4.—On opening the chest it was found that both lungs exhibited on the upper posterior lobes numerous petechiæ under the pulmonary pleura, the peripheral lobules of these parts being much congested. There were numerous adhesions by recent soft lymph between the lower lobes of the lung and the costal pleura, particularly laterally. In the liver there were several

reddish streaks and patches reaching from the surface of the organ to a depth of about a quarter of an inch. In these patches the liver tissue was much softened. The spleen and kidneys, with the exception of slight congestion, appeared normal. In the placenta there were numerous petechiæ.

Lung.—Sections made through the portions above mentioned as containing the congested lobules showed not only great congestion of the blood-vessels, large and small, but a large amount of hæmorrhage; blood in substance was present in the air vesicles and infundibula, in the lymph spaces of the interlobular septa, and in the tissue and lymphatics of the pleura.

Liver.—Under the capsule, as well as in the substance of the liver, there occurred, in connection with the interlobular branches of the portal vein, larger and smaller foci of inflammation consisting of numerous round-cells. Some of these foci were several millimetres in diameter, others were very small. From the interlobular tissue the inflammation extended into the lobules between the liver-cells. The liver-cells of these lobules involved in the inflammatory process were swollen up, and many of them undergoing disintegration. In some of these foci, particularly those situated in the vicinity of the capsule, the round-cells were so much crowded as to give these foci an appearance almost like miliary abscesses. The blood-vessels were much distended and filled with blood.

Kidney.—Sections showed well-marked glomerulo-nephritis; infiltration of the sheath of the cortical arterioles, with numerous round-cells. The epithelium of the convoluted tubules was swollen, opaque, and in many places disintegrating.

Cow No. 3.—In the lungs there were numerous lobules, especially in the peripheral parts, which showed great congestion; there were, in addition, pleural adhesions, the cortex of the kidney was congested, but its medulla was pale.

The lungs and kidneys showed, on microscopic examination, the same appearances as in Cow No. 4; in addition, there was a good deal of round-celled infiltration in the walls of the infundibula and bronchi in the lung, and round the arterioles in the kidney.

In sections of the ulcers on the teats the corium was found to be infiltrated throughout the whole extent of the ulcer with round-cells. In the superficial layers of the stratum Malpighi, close to the stratum lucidum, as also in the stratum lucidum itself, there were

numerous cavities of different sizes. These cavities lay side by side, the most superficial ones being covered by the stratum lucidum, or extending between the layers of this stratum.

At the marginal parts the cavities, although placed side by side, were well separated from one another by thicker or thinner trabeculæ, composed of epithelium, while at or near the centre of the ulcer these trabeculæ were destroyed, and the cavities had become confluent, and the covering layers of the cuticle having here also given way, their contents extended on to the free surface of the ulcer. In short, Dr. Klein states that all the anatomical details of the distribution and arrangement of these cavities recalled vividly the conditions observed in the vesicles of cow-pox.

In the discharge from the ulcers micrococci, diplococci, and short chains were met with. In sections of the ulcer, stained to show the micro-organisms, diplococci and chains were found in the contents of the superficial cavities, and in the depth of the epithelium, and in the crust, and in the necrotic parts of the ulcer numerous clumps or zooglœa of micrococci.

As a result of this investigation, Dr. Klein concluded that the cow disease at Hendon was scarlet fever in the cow, a disease as yet unknown to the veterinary profession. That thus the danger of scarlatinal affection from the disease in the cow was real, and that towards the study and supervision of this cow disease all effort ought to be directed in order to check the spread of scarlet fever in man. Further, the fact of the disease being unknown and unrecognised by veterinary surgeons should stimulate them to try and recognise it.

Outbreaks of a disease of the teats of cows alleged to be identical with the cow disease at Hendon were reported to Professor Brown. I had an opportunity of inspecting a few cases from these outbreaks. They presented brown or reddish crusts on the teat, which, when picked off, exposed a granulating thickened base, with exudation of a lymph-like discharge and slight hæmorrhage.[1] Traces of an eruption of the skin were noticed in one, patches

[1] The appearances of this stage of the eruption in these cases corresponded with the description of the naked-eye appearances of the Hendon cow disease. I have had an opportunity of studying a pustular disease of the udder and teats, and the so-called Edinburgh cow disease, and other eruptions of the udder and teats, which were quite distinct from the cases which corresponded with the Hendon cow disease.

devoid of hair,[1] and running from the eyes in others. These symptoms could only be regarded as coincident conditions. One of the cows was suckling a calf, and this calf had previously been suckled by another cow also affected with the same eruption of the udders and teats. The calf had developed flat, angry-looking ulcers on the nose and hard palate, and an eruption in the hairy part of the nose near the muco-cutaneous line of union.

I was anxious to enter fully into the history of these cases, to question the milkers, to ascertain if the disease was communicated to their hands, and to obtain all the information possible that would throw light on the pathology of the disease. But permission for me to visit the farm could not be obtained. Fortunately, another outbreak of an eruptive disease of the teats was reported, which was also alleged to be identical with the so-called Hendon cow disease. This outbreak was raging in some farms in Wiltshire. In this case every facility was given by the owner of the estate for a thorough investigation into the disease. Not only were animals sent from the farm to the Royal Veterinary College, but I was allowed to visit the farms to inspect all the affected animals, and to make every investigation, with the hearty co-operation of the bailiff of the farms, and the voluntary assistance of the head cowmen and those under them. Some of these cowmen were not only unusually intelligent, but also had had an experience of cows for more than half a century. Thus, I had not only every opportunity for studying the disease on the lines indicated by Dr. Klein, but I was also able by repeated visits to the farms to enter into the clinical history of the disease in the cow, to study very fully the nature of the disease on the hands of the milkers, and to trace the probable mode of introduction of the disease on the estate, and the way in which it spread from one part of the herd to another.

Account of an Investigation into the Micro-pathology of a Disease of Cows in Wiltshire, and its connection, if any, with Scarlet Fever in Man.

I will now proceed to give the details, step by step, of my investigation into the eruptive disease of the teats of cows in Wiltshire.

My first observations were made upon a cow which had been

[1] "When cows are too much confined or kept on improper diet, the hair frequently falls off, leaving bald patches" (Willan).

purchased and removed to the Royal Veterinary College. This cow I shall speak of as No. 1. She was fairly well nourished, ate well, and was reported by the attendant to be normal in her milk supply. Her temperature was normal on admission, but subsequently rose from mammitis occurring as a complication. There was no desquamation or eruption on the skin, or any patches destitute of hair.[1] But there was a characteristic disease of the teats and the udder between the teats. On the right teats there were numerous sores, covered with crusts varying in size from a pea to a penny. The crusts varied in thickness, and were generally fissured. In some they were flat, in others conical, some were with difficulty removed with forceps, others were readily detached. The crusts varied in colour from reddish brown to a very dark brown or almost black. On detaching or scraping a crust there was a granulating and somewhat indurated base. On the right anterior teat there were several ulcers, from which apparently the thick crusts had been detached, and new scabs were forming. On the left posterior teat there were unusually large, dark brown or blackish crusts, covering a very extensive area of ulceration, extending over the whole of the lower third of the teat.

In another cow, also conveyed to the Royal Veterinary College from Wiltshire (which I shall speak of as No. 2), there was the same disease on the teats. But the condition of the teats was not nearly so severe. The sores on the teats were covered with thick crusts, but though varying in size they were more regular in form, and more circumscribed.[2]

Having entirely removed the crusts from some of the ulcers of cow No. 1, I squeezed the base, and from the discharge I made a number of inoculations in nutrient gelatine and nutrient agar-agar. I also collected the discharge in capillary tubes and sealed them immediately, and also made a number of cover-glass preparations. These were stained in the ordinary way with fuchsine or methyl-

[1] This was probably due to the excellent hygienic conditions of the Wiltshire farms.

[2] At subsequent visits to the farms I was able to study a great number of cases at different stages of the eruption. The history of the vesiculo-pustular eruption (cow-pox) from which this extensive ulceration and scabbing was derived as the result of the manipulation of the milkers, will be published in full when the investigation is completed (see 'Brit. Med. Journ.,' July 7th and 14th, 1888).

violet, and showed here and there micrococci singly, in twos, or in fours, or a few cocci linked together. Cover-glass preparations from a scraping of a crust showed the presence of micrococci in greater numbers. The inoculations had been made for the most part in tubes of nutrient gelatine and nutrient agar-agar solidified obliquely. To separate the different organisms that might be present in the discharge from the ulcers[1] I employed either Koch's plate-cultivations or Esmarck's method, or I simply drew the platinum needle in a line on the surface of the nutrient medium. Agar-agar tubes inoculated in this way were placed in the incubator at 37° C, and gelatine tubes were kept at about 18° C. In the course of two or three days in the tubes of nutrient agar-agar placed in the incubator a growth commenced along the track of the needle, consisting mostly of china-white, yellow, orange, and little dot-like colonies. From these various colonies cultivations were established in nutrient gelatine. In this way pure cultivations were obtained of seven different micro-organisms. The china-white, yellow, and orange growths were found to correspond with the micrococci named *Staphylococcus cereus albus*, *Staphylococcus pyogenes citreus*, and *Staphylococcus pyogenes aureus*. The dot-like colonies with darker centres were composed of micrococci, which became the subject of the closest study (Plates XXIV and XXV).

The individual cocci are small spherical cells, with a special tendency after fission for the resulting elements to remain attached to each other, forming chains or rosaries. There may be a few, three or four, elements linked together, or a number of individuals forming straight or serpentine chains. The size of the individual elements is not always equal, here and there in a chain a larger element appearing. In plate cultivations the appearances of the colonies are not very striking. They appear to the naked eye after three or four days as extremely minute, greyish-white, translucent dots, which under the microscope have a slightly yellowish-brown colour. They are finely granular and well defined. They do not liquefy the gelatine, and after several weeks do not exceed the size of a pin's head. If the surface of nutrient gelatine solidified obliquely is traced over once or twice with a platinum needle bent at the extremity into a little hook charged with the micrococci, a

[1] Cultivations were also made from pus, from crusts, and from the milk of different cases. Altogether nearly 300 cultures and subcultures were prepared, many of which had to be abandoned to afford time for other lines of inquiry.

DESCRIPTION OF PLATE XXIV.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

A streptococcus from the ulcers on the teats. From a preparation made from the broth which collects at the bottom of freshly prepared nutrient agar-agar. In this case the nutrient agar-agar was a special kind made with Brand's extract of meat according to Dr. Klein's receipt. The growth was examined after two days at 37° C. × 900.

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Fig 1

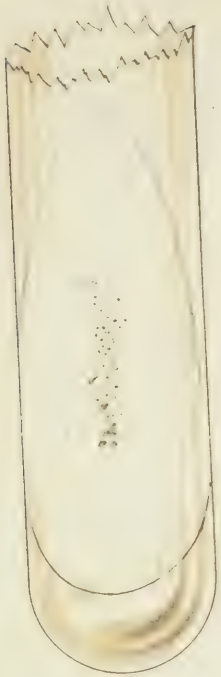


Fig 2

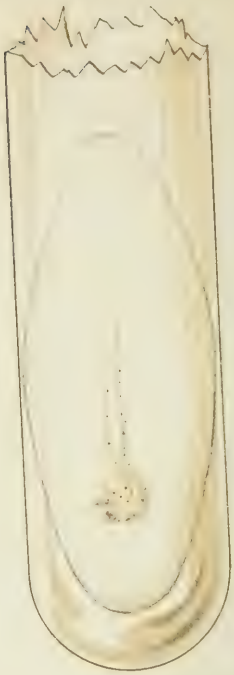
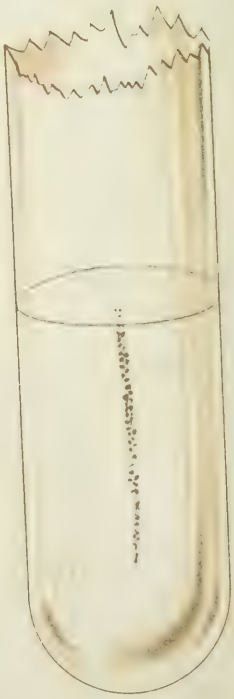


Fig 3

Fig 4



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DESCRIPTION OF PLATE XXV.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From drawings by himself.

FIG. 1.—Growth of a streptococcus on the surface of nutrient gelatine solidified obliquely. After four days at 18° C.

FIG. 2.—A similar cultivation after four months at 18° C.

FIG. 3.—Growth of the same streptococcus on the surface of nutrient agar-agar after four days at 37° C.

FIG. 4.—Growth, after four months, in the track of the inoculating needle introduced into the depth of the nutrient gelatine.

The cultivations are represented, as they appear by reflected light, under slight magnification with a pocket lens.

ribbon-shaped film develops in two or three days. This film is composed of minute, greyish-white, translucent dots or droplets, which can be more easily recognised with the aid of a pocket lens. According to the number of organisms sown on the jelly the dots or colonies may be completely isolated, or form a more or less continuous film. The film by reflected light has an iridescent appearance like mother-of-pearl, but has a bluish or bluish-grey tint by transmitted light, and with a pocket lens appears distinctly brownish. The gelatine is not liquefied, and even after several weeks the cultivation is limited to the inoculated area, and the individual colonies are not larger than pins' heads. In gelatine cultivations of the same age, but kept in the incubator at 18° C., the colonies get irregular in form, especially at the margin of the film, and give the growth an arborescent, fringed, or serrated appearance. Cultivated on the oblique surface of nutrient agar-agar at 37° C. the growth is very similar, forming a film composed of minute dot-like colonies like grains of sand. But the film appears less transparent, is whiter, and the colonies have a greater tendency to get irregular in form. If inoculated with one tracing of the needle the growth is scanty but tends to get thicker in the centre than towards the margins, which may have a terraced appearance. Inoculated in the depth of gelatine, there appears after a day or two at 18° C. a thread-like growth along the track of the inoculating needle. This delicate growth is found on examination with a pocket lens to consist of a linear series of extremely minute granules. In a few days more the beads or granules become more marked, but even after weeks the cultivation only appears like a string of minute white, compact, globular masses or grains. In broth at 37° C. the cocci in twenty-four hours create a turbidity, and gradually develop chains varying in length according to the age of the cultivations. Even in forty-eight hours there may be chains of eight, ten, or twenty elements. After a few days the growth settles down at the bottom of the tube in the form of a white deposit, while the supernatant liquid becomes clear again. Cultivated in milk at 37° C. they convert it in two or three days into a solid mass, while uninoculated control tubes remain liquid. Inoculated subcutaneously in the ear of rabbits they produce in two days an inflammatory thickening with erysipelatous redness and without suppuration. Thus the morphological and cultural characters and effect on the rabbit's ear of this streptococcus are identical with the description given by Rosenbach

and others of the morphological and cultural characters of the micro-organism found by Ogston in suppurations in man and called by the former *Streptococcus pyogenes*. I am therefore led to regard this streptococcus as belonging to the same species as the streptococcus isolated from man. Moreover, the organisms with which it was associated in the discharge from the ulcer correspond with the organisms which are commonly present in pus from a human source.

On Saturday, November 26th, I paid my first visit to the farms from which the diseased cows Nos. 1 and 2 had been purchased. On my arrival I made inquiries as to the first occurrence of the outbreak. I gathered that it appeared about the end of September, but it was said that no fresh cows were imported at that time. The disease commenced at a farm which I shall call V. The cows were changed about from farm to farm, so the disease spread through all the farms W, X, Y, Z, the disease being conveyed from diseased to healthy cows by the hand of the milker. In all there were about 160 cows.

From the first two milkers I encountered I took down the following history. They said that the teats of the cows swelled, were hot to the touch, and very tender; as a result the animals were milked with difficulty. In milking some of the severer cases the milkers resorted to the standing position, as otherwise they and their milk-pails were upset. In some cases the pain caused by milking was so great that the animals became almost unmanageable, and had to be tied by the legs. One man described the pain caused by milking the severe cases as "most barbarous."

The account which these men gave me of the eruption was as follows: They said a little white bladder would rise up on the teat, which would become as large as a pea or much larger, and then was burst in the milking. As the result a sore formed and became covered with a scab. "Matter" formed under the scab, and as the scabs were broken in milking "matter" and blood would come out on their fingers and get mixed with the milk. Sometimes the scabs would become rubbed off, in other cases they fell off, and finally in four or five weeks the sores would be healed. I was, later on in the day, an eyewitness of the milking operations, and can therefore bear testimony to the accuracy of the description of the milkers of the severity of the treatment, and of the contamination of the milk which resulted. They further supplemented their description by asserting that the disease was cow-pox, a disease which they said

had existed in Wiltshire in the time of their fathers and forefathers.

At these farms I found that the disease had been communicated to several of the milkers. They had been inoculated some weeks before my visit; I had therefore to rely a great deal on the description given by the men.[1] I will describe some of the cases. Cowman R— showed me a scar on his right hand. He said he was the first to catch the disease. He described it as coming as a hard painful spot which formed "matter" and then a "big scab." He had continued milking ever since, but had no fresh "places." Cowman F— about a month ago noticed spots which appeared on both hands. His fingers swelled and were painful. He says it came first like a pimple and felt hard. Then it "weeped out water" in four or five days. There were, he says, red marks creeping up his arm and a sort of throbbing pain, and he could not sleep at night. I found on the right hand a scar and on the left hand an ulcer about the size of a shilling covered with a thick black crust. The crust was partially detached and exposed a granulating ulcer. It was the exact counterpart of an ulcer on a cow's teat.

Cowboy H— had both hands bad about a month ago; first the index finger of the left hand, and then the right hand. He says that it came up like a hard pimple, and the finger became swollen and red. After a few days it "weeped out water," and then matter came away. Both his arms were swollen, but his left arm was the worst. About a fortnight after he noticed "kernels" in his armpits, which were painful and kept him awake at night. His arms became worse, he could not raise them, and he had to give up milking. He also had inoculated himself on the lower lip. On examination, I found that the axillary glands were still enlarged and tender. He volunteered the statement that the places were just like the sore teats. H—, the bailiff's son, had a sore on the upper lid of the right eye, and on his left hand. In both places he had previously been scratched by a cat. The right hand had also been infected. It commenced, he says, a fortnight

[1] On the occasion of subsequent visits to the farm, I had the opportunity of studying fresh cases. Eight out of eleven milkers had vesicles on their hands. I was able to watch the course of the eruption in a lad removed to London. These cases will be published in full in connection with my further inquiries as to the nature of this disease (see 'Brit. Med. Journ.,' July 7th and 14th, 1888).

ago. His hands were swollen, red, and hot. He felt very poorly and went to bed. Little spots like white blisters appeared on the back of his right hand, these broke and "rose up" in thick scabs. He was very ill for two or three days, but did not send for a doctor. He had painful lumps at the bend of the arm and in the armpit. He gave up milking and had not taken to it since.

I now pass on to describe some experiments undertaken with a view to studying the transmissibility of the disease.

With the discharge and with scrapings from the ulcers on the teats of cow No. 1, I inoculated four white mice, one guinea-pig, and two calves.

In the white mice and guinea-pig inoculated subcutaneously with a small quantity of the lymph-like discharge no result followed.

Of the two calves, one, a large calf, which I will call A., was inoculated by scarification in both ears; the other, a small calf, which I will call B., was scarified in the left ear. Scrapings from the ulcers were rubbed into the places thus prepared. In addition, in the small calf an incision was made through the corium in the left groin, scrapings from different ulcers on the teats were well rubbed in with the blade of the scalpel, and a portion of crust inserted into a small pocket in the subcutaneous tissue. In the ears and the groin there were positive results. In the large brown calf one of two places inoculated in the right ear went through the following changes: On the third day there was apparent vesiculation and commencing formation of crust. From day to day the crust thickened, and on the eighth day the crust was at its height and detached at its edges. By removing the scab an ulcer was exposed; there was slight inflammatory thickening. About the thirteenth day the ulcer had quite healed.

Very similar appearances resulted in the ear of the smaller calf. The result of inoculation in the groin was of a very much severer character. In the course of two or three days the incision had apparently commenced to heal by scabbing, but there was an area around which was inflamed and painful on manipulation. The inflammatory thickening which resulted continued to increase around the seat of inoculation, and the thickening could be felt to extend deeply in the groin. Suppuration followed, and on firm pressure pus welled up through the wound. The wound then showed very little disposition to heal, and the calf began to exhibit

marked constitutional symptoms. During the second week after inoculation the animal became very dull, and was reported by the attendant as refusing to feed.

Diarrhœa supervened and lasted for several days, and bloody urine was passed.

The calf was also noticed to cough, and the cough gradually increased in severity. Thirty-six days after the date of inoculation it was decided to kill the calf and examine the condition of the viscera. The *post-mortem* was made under the supervision of Professor Brown and Professor Axe. I have already submitted the microscopical specimens for examination, and I will now proceed to describe the results which were found at the *post-mortem*, and the appearances which will be found in the microscopical preparations.

Lungs.—Upper and middle lobes of each lung were adherent to the walls of the chest; there was patchy congestion, especially of the middle lobe, and patches of recent adherent lymph. Posterior parts of the upper lobes of both lungs were completely consolidated, and on section varied in colour from brick red to greyish white. The interlobular tissue was infiltrated with inflammatory products, which mapped out the tissue of the lung in small indurated areas, in which the tissue was granular looking and friable. These appearances in the upper lobes were due to pleuro-pneumonia. Scattered through the other lobes of both lungs were white, mostly firm, nodules raised above the level of the surface of the lung. They were surrounded by a zone of congestion, and in some cases sections were composed of indurated, in others of friable, lung tissue. In the posterior part of the right upper lobe there was a recent infarct.

Bronchial glands.—The bronchial glands at the roots of each lung were enlarged to two or three times their natural size, and were firm and hard on section.

Pericardium.—The parietal surface of the pericardium was covered with recent adherent lymph. The visceral surface of the pericardium was normal.

Aorta.—Along the external surface of the aorta were chains of enlarged lymphatic glands connected by dilated lymphatic vessels. These glands were dark red or purplish in colour, from hæmorrhage into their substance.

Heart.—The heart was normal, and the endocardium not stained.

Œsophagus.—There were chains of red glands on the œsophagus similar to those along the aorta.

Mesenteric glands.—The appearance of the mesenteric glands was very striking. The mesentery was dotted along the lymphatic vessels with glands, varying in size from a large shot to a pea, which were deep red or prune coloured. In addition, there were here and there enlarged glands without hæmorrhage into their substance, and greyish in colour.

Spleen.—There were scattered petechiæ on the spleen.

Kidneys.—The kidneys were firm on section, and there was marked congestion in both, while it was more pronounced in one kidney than the other.

Liver.—The liver was congested, the congestion being more marked in patches.

Lung. — Sections from the consolidated upper lobes showed under the microscope thickening of the pleura and infiltration with round-cells. The exudation filled the alveoli, and was breaking down in some cases in the centre. The vessels were injected, and there were hæmorrhages into the alveoli. The periphery of the lobules was infiltrated with round-cells.

Kidneys.—Slight infiltration around glomeruli and arterioles with round-cells; epithelium with convoluted tubes, granular and disintegrating; hæmorrhage in straight tubes, and engorgement of vessels. (Plate XXVI, figs. 1 and 2.)

Liver.—Inter- and intra-lobular vessels engorged; intralobular collections of round-cells displacing the liver cells, interlobular connective tissue infiltrated with round-cells; liver-cells granular and cloudy. (Plate XXVII.)

There can be no doubt from the symptoms and *post-mortem* appearances that this calf suffered from septicæmia, as the result of introducing the septic virus and crust subcutaneously in the groin; moreover, it is obvious that there was a predisposition to septic poisoning in this case, owing to the lowered vitality induced by the concomitant attack of pleuro-pneumonia.

The two Wiltshire cows, which I have referred to as Nos. 1 and 2, were sent for *post-mortem* examination to be made at the Brown Institution; so that Dr. Klein might give his opinion as to whether any *post-mortem* appearances which might be present were similar to those he had seen in two cows from Hendon. A day having been fixed by Dr. Klein for the *post-mortem* examination, I accom-

DESCRIPTION OF PLATE XXVI.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From drawings by himself.

From a section of the kidney of a calf in which septicæmia was produced by inoculation with scrapings from the ulcers of a Wiltshire cow.

FIG. 1.—*a*. Malpighian corpuscle with glomerulo-nephritis. *b*. Small collection of round-cells. × 230.

FIG. 2.—Straight tubes with hæmorrhages. × 230.

Fig 1

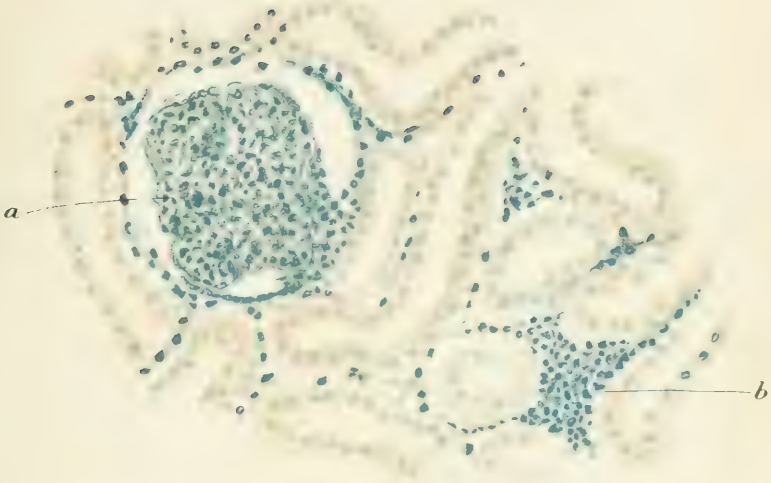
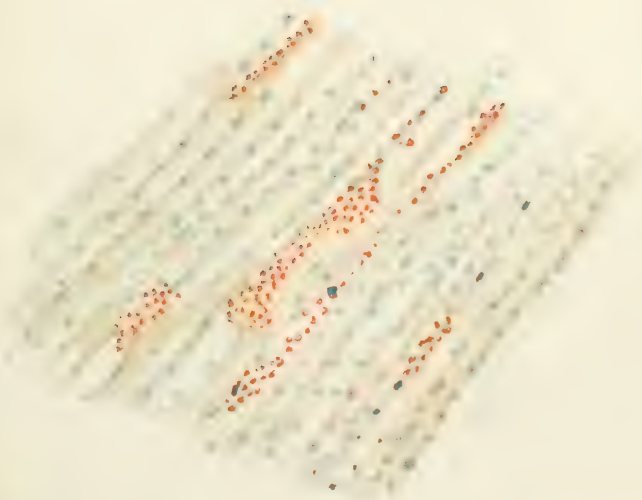


Fig. 2.



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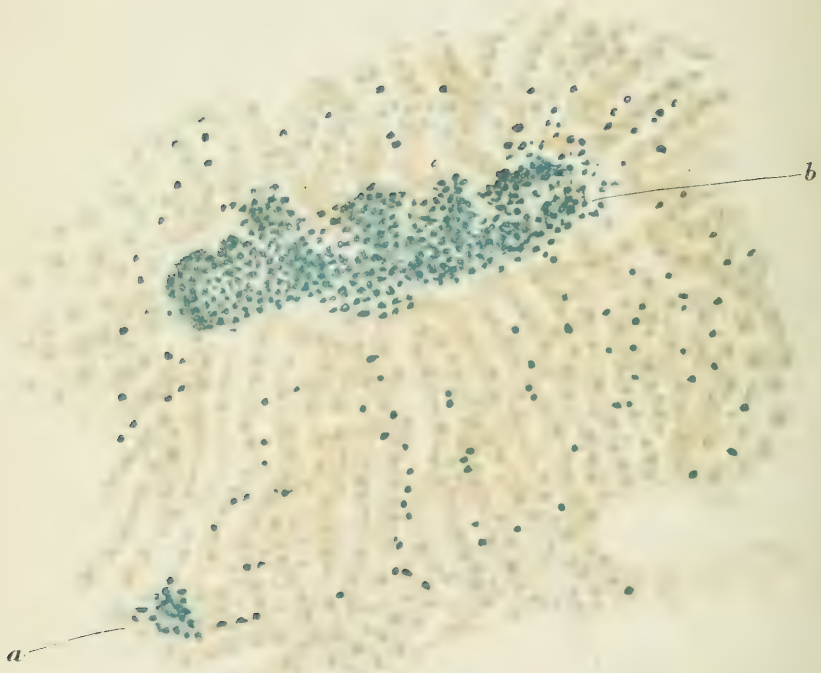

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DESCRIPTION OF PLATE XXVII.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

From a section of the liver of a calf in which septicæmia was produced by inoculation with material from the ulcers on the teats. × 230.

Between the liver-cells, with their faintly stained nuclei, are numerous round-cells.

At (*a*) is a small collection of round-cells, and at (*b*) a much larger one amounting almost to a miliary abscess.

Fig 1

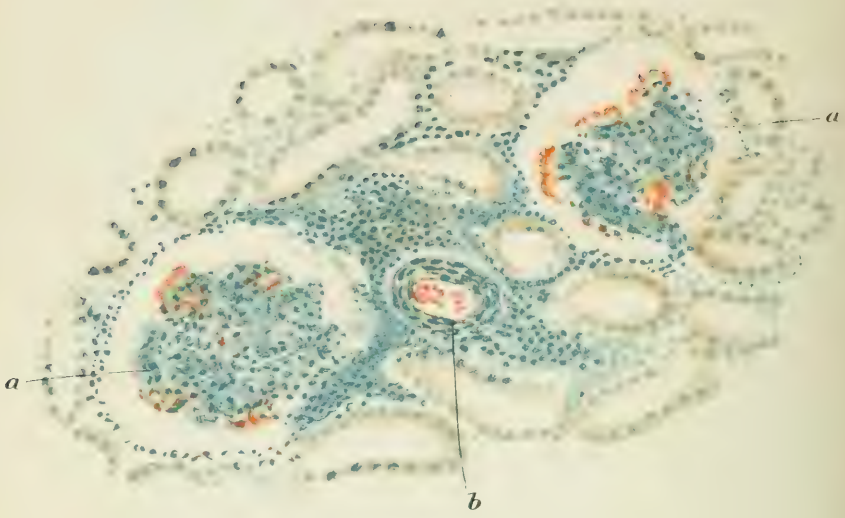
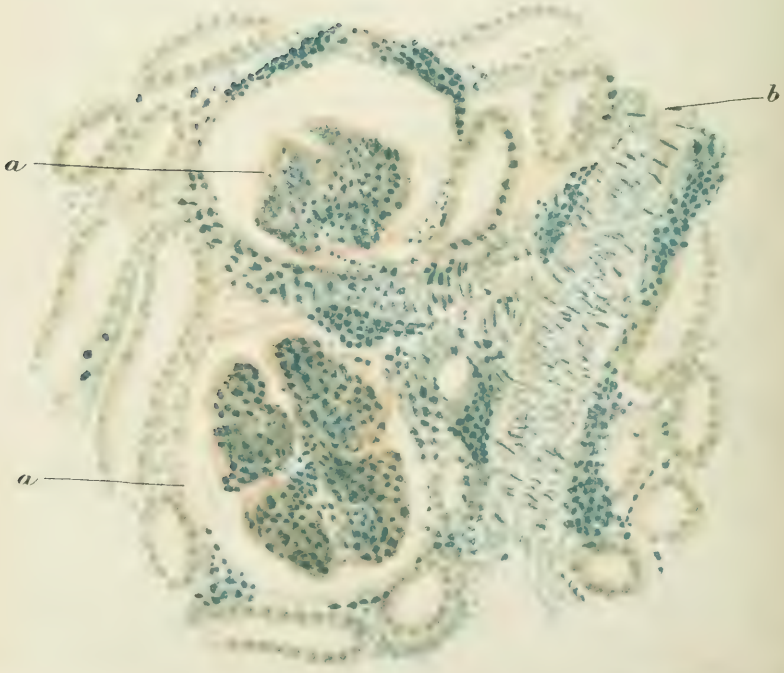


Fig. 2.



Edgar Crookshank del.

Fred^k Dangerfield Lith London 1890

DESCRIPTION OF PLATE XXVIII.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From drawings by himself.

FIG. 1.—From a section through the cortex of the kidney of cow No. 1. × 230.

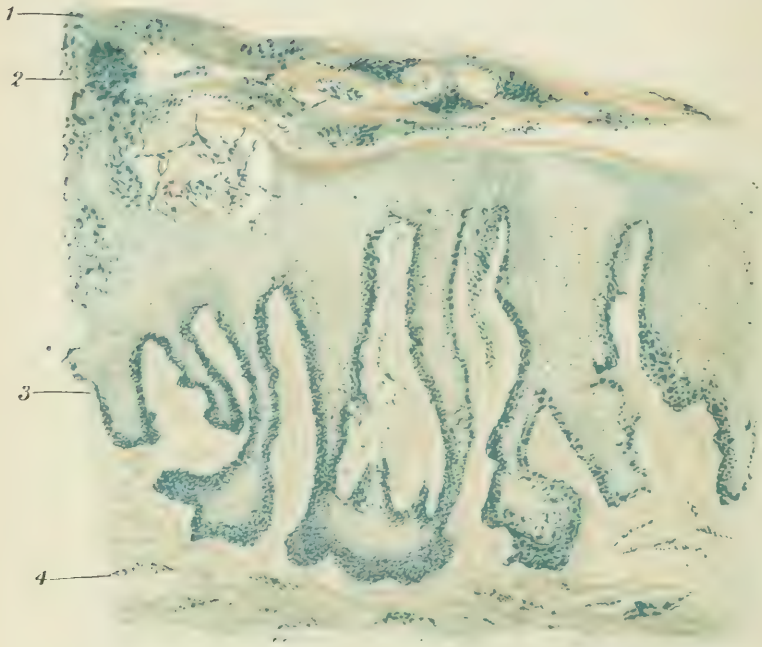
a. Malpighian corpuscles, showing well-marked glomerulo-nephritis, and hæmorrhages.

b. Section of an arteriole surrounded with round-cells.

FIG. 2.—From another part of the same section. × 230.

a. Malpighian corpuscles showing well-marked glomerulo-nephritis.

b. An arteriole surrounded with collections of round-cells.



Edgar Crookshank del

Fred^k Dangerfield Lith London 1620

DESCRIPTION OF PLATE XXIX.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

Vertical section of the peripheral part of an ulcer on the teat of cow No. 2. × 90.

1. Horny layer.
2. Superficial layer of the stratum Malpighii with cavities filled with round-cells.
3. Deep layer of the stratum Malpighii infiltrated with round-cells.
4. Superficial corium infiltrated with round-cells.

panied Professor Brown to see the result. Inasmuch as there was no suspicion of any visceral disease complicating the eruptive disease of the teats, it was not surprising to find that the naked-eye appearances at the *post-mortem* were practically negative. All that could be noted of the viscera was as follows: petechiæ on the spleen and liver, a few whitish irregular spots, fatty patches, on the surface of the cortex of the kidney. I have already mentioned that there was an unusual condition of ulceration in this cow, and milking had become a matter of great difficulty in inexperienced hands. The result was that soon after the cow had been at the Royal Veterinary College the udder became enlarged, and the teats red, tense, swollen, and œdematous. At the *post-mortem* an incision into the udder revealed an enormous abscess.

At the *post-mortem* of cow No. 2 there was a somewhat similar condition, and the visceral changes were negative. Though the naked-eye appearances in cow No. 1 were practically negative, the result of examining sections of the kidney under the microscope were extremely instructive and interesting, as they showed that marked changes had taken place which were indicative of septic complication.

The sections showed glomerulo-nephritis; there was infiltration round the capsule of Bowman with round-cells; there was infiltration also of the sheaths of the vessels with round-cells, especially of the cortex. The blood-vessels in the boundary zone of the medulla were engorged, the arterioles of the glomeruli were also engorged, and there were slight hæmorrhages into the capsule. The epithelium of the convoluted tubes was granular, opaque, and in some parts breaking down. (Plate XXVIII, figs. 1 and 2.)

Sections of the ulcers of the teats of these cows were also carefully examined.

Sections of a portion of the spreading ulceration of the teat of cow No 1 showed only a superficial ulceration not extending far into the depth of the tissue. There were marked hæmorrhages in the corium. These appearances differed from the appearances in sections of a circumscribed ulcer from the teat of cow No. 2, in that in sections of the latter the ulceration extended much more deeply into the tissue, and there was present a characteristic cellular character. (Plate XXIX.) These spaces or cavities existed in the stratum Malpighi, especially in the superficial layers. They were observed to be either empty or filled with granular matter and

round-cells, and were separated from one another by trabeculæ. In both cases the corium was infiltrated with round-cells. The appearances, in short, of sections of the more circumscribed ulcers of cow No. 2 corresponded in a remarkable degree with the anatomical features which are described as characteristic of the structure of the eruption in cow-pox. By staining sections from these ulcers by the method of Gram without a contrast stain, an invasion of the ulcerated tissue by micrococci was very beautifully demonstrated. In the crust and necrotic portion of the ulcer there were found masses of staphylococci and isolated micrococci, diplococci, and short chains. Micrococci in twos, fours, and short chains of three or four elements, were also found here and there extending into the depth of the epithelium, and a cellular space in a section was found almost completely occupied by a streptococcus many times convoluted or twisted on itself.

There were altogether about 160 cows on these farms. Only a few had proved refractory, and had not taken the disease at all. The rest had contracted the disease in varying degrees of severity. About fifty at a time were dry, and they escaped until they were in milk again. The milk was drunk on the farms and in the village, but most of it, amounting to eight churns a day, that is sixty-four barn gallons or 1088 pints, was sent twice a day to a large city. Not being distributed in a wealthy neighbourhood we may roughly estimate the number of persons drinking the milk at about 1500 or 2000. Most careful inquiries were instituted to ascertain the existence of scarlatina among consumers of the milk. So far the research was completely analogous to the Hendon investigation, and the contamination of the milk, which I had myself witnessed, led me to so fully believe in the probability of scarlet fever being found among the consumers of the milk, that I would not be satisfied until I had obtained the most reliable evidence that such was really not the case. Under the circumstances it was a matter of surprise that no cases of scarlatina were found either on the farms or in the village, and that in the town district in which the milk was distributed there was no epidemic produced by its use.

It is obvious that farmers, dairymen, medical men, and indeed experts in the diseases of cattle, would be liable to pronounce an outbreak of the Wiltshire disease to be the alleged bovine-scarlatina.

In studying this disease, therefore, we must place ourselves in

the position of endeavouring to discover what conditions may exist which will enable the skilled practitioner to distinguish an outbreak similar to the Wiltshire disease from an outbreak of bovine-scarlatina.

For it is certain that this Wiltshire disease could not possibly be regarded as another instance of bovine-scarlatina, as no scarlatina resulted from the use of the contaminated milk.

This naturally led me to reflect upon the immense importance of ascertaining some means of differential diagnosis. For, if during the Wiltshire outbreak *as a mere coincidence* there had happened to be cases of scarlatina among the consumers of the milk, and this condition of the cows had been discovered by an inspector, the consequences would have been disastrous to the dairy farmer.[1] But if we endeavour to find differences we are led step by step to the conclusion that there is no means at our command of distinguishing this disease from the description which has been published of the clinical characters and the pathology of the disease which was observed at Hendon.

In both cases we meet with a disease which spreads through a whole herd of milch cows.

In both cases the disease is characterised by the appearance of vesicles, which are broken by the hand of the milker, and irritated into deep ulcerations.

In both cases the disease is conveyed from one cow to another by the hand of the milker.

In both cases the vesicular eruption is communicable to the hand of the milker.

In both cases the disease is a mild, not a fatal one, and if a cow were killed for diagnostic purposes no assistance would be gained, as in both cases the *post-mortem* appearances which may be found are not to be distinguished from accidental complications.

In both cases the naked-eye appearances and the duration of the ulcers of the teats are the same.

[1] "The unfortunate farmer in whose herd this disease has recently occurred must have lost upwards of £1000 by the loss of milk and depreciation in the value of his stock" (Dr. Cameron on the Hendon cow disease). "The farmer . . . obeyed the suggestions that were made to him during the inquiry, although he did not always see the reason for them, and although they interfered to a dismal degree with his immediate trade interests" (Mr. Power on the Hendon cow disease).

In both cases sections of the ulcers show under the microscope identical appearances of a characteristic cellular character.

In both cases the tissue of the ulcer is invaded by micrococci, singly, in pairs, and in chains.

In both cases from the purulent discharge of the ulcers streptococci may be cultivated, which are identical in the description of their form and characters on cultivation.

In both cases the same results are produced by inoculation of calves with septic virus.

As I was thus unable to establish any difference, I was naturally led to believe in the possibility of the identity of these two affections, and therefore, as a result, to question the possibility of scarlatina in man having arisen from the disease in the cows at Hendon.

But whether we accept the identity of the two diseases or not, there inevitably arises the possibility of the results obtained in the investigation of the Hendon cow disease admitting of another interpretation by the light thrown upon the matter by the pathological researches embodied in the Wiltshire investigation.

An unbiassed investigator is at once forced to the conclusion that this is the case.

Thus, if we examine the chain of argument which has been brought forward to maintain the existence of cow-scarlatina at Hendon, we find that it is urged:

(1) That the Hendon cow disease was a disease in which the *post-mortem* appearances resembled scarlatina.

(2) That this disease was associated with a streptococcus, which produced by inoculation in calves a disease with *post-mortem* appearances similar to those of the Hendon cows.

(3) That a streptococcus regarded as identical with the one above mentioned was found in certain cases of scarlatina in man, which when inoculated in calves produced *post-mortem* appearances similar to the *post-mortem* appearances in the original Hendon cows and in certain cases of scarlatina in man.

We must here consider what are the *post-mortem* appearances in scarlet fever?

"On *post-mortem* examination of scarlatinal patients most internal organs appear to the naked eye fairly healthy. The liver and kidneys may be somewhat softer than usual, and the blood imperfectly coagulated. In so-called malignant cases there may be collapse and hypostatic congestion of the lungs, and hæmorrhage

into and at the surface of internal organs. The throat generally presents distinct traces of inflammation and ulceration. The solitary intestinal glands and Peyer's patches are somewhat enlarged. The only other morbid appearances (and they are sufficiently important) are such as are connected with the sequelæ and complications of the disease. *These, however, though common to scarlet fever, are not peculiar to it.*" (Bristowe.)

In illustration of the last statement I may draw attention to the fact that the microscopical appearances of the kidney of the Wiltshire cow are identical with those which were regarded as indicating scarlatina in the Hendon cow (cow No. 4). (See Plate XXVIII.)

Indeed, the statements as to the *post-mortem* appearances in the Hendon cows, when studied, not only do not necessarily indicate scarlatina, but they cannot even be considered of primary importance, or, indeed, as throwing much light on the question of scarlatina at all. The description of the naked-eye appearances in both cows only suggests coincident pleurisy or pleurisy with pneumonia. The microscopical appearances in both are, as I have already said suggestive of septic complication.

A careful examination of the *post-mortem* appearances of calves inoculated with scraping of an ulcer of a Hendon cow, or with cultivations of the streptococcus from certain cases of scarlatina, brings to light much more striking changes. These appearances, however, cannot be regarded as indicative of scarlatina. They are in reality the *post-mortem* appearances of septic poisoning, and common therefore to many diseases. This is clearly shown by comparing the *post-mortem* appearances in the calf which was killed while suffering from septicæmia, as the result of inoculation from the ulcers of a Wiltshire cow. These visceral changes, excluding the concomitant pleuro-pneumonia, are not to be distinguished from the *post-mortem* appearances described in the calves inoculated by Dr. Klein. Consequently, that the streptococcus found in certain cases of scarlet fever should produce on inoculation in calves certain *post-mortem* appearances which are common to many diseases, and should fail to produce fever, ulceration of the tonsils, or scarlatinal rash, or any condition in the least resembling clinically the disease in man, and yet that the result should be regarded as scarlatina in the calf, is a conclusion quite untenable.

It is true that visceral lesions similar in character may be produced in calves whether they be inoculated with scrapings from ulcers, or with streptococci from ulcers, of the Hendon cow disease, or with streptococci from certain cases of scarlet fever. In both cases the streptococcus is a septic microbe, and inoculation of this microbe, or the inoculation of septic virus, is liable to produce septicæmia. *These facts constitute a mass of evidence which justifies the conviction that the pathological data which lent support to the theory that the vesicular disease of the teats of cows at Hendon was scarlatina in the cow, admit of an entirely different interpretation, and, consequently, the theory that the Hendon milk was contaminated with the virus of scarlet fever from some human source which Mr. Power failed to discover, is not invalidated by the researches of Dr. Klein.*

On a Streptococcus alleged to be the Contagium of Scarlet Fever.

In support of the theory that the Hendon cow disease was scarlatina in the cow, it is alleged that a similar microbe was discovered in human scarlet fever which produced on inoculation in calves a disease which was interpreted as the Hendon cow disease, and as similar to scarlet fever in man.

This streptococcus was found by Dr. Klein in five out of eleven cases of scarlet fever in man, twice in association with certain other micro-organisms, and three times alone. The micro-organisms were isolated by inoculating tubes of nutrient gelatine, solidified obliquely by streaking the surface with blood taken from the finger, the arm, or the heart after death. These investigations were carried out during 1886–87.

Those cases from which the organism was obtained were all cases with *ulcerated throat*, and the culture experiments were made at or about the day *at which the temperature was at its maximum.*

Dr. Klein regarded this streptococcus as the contagium, virus, or actual cause of scarlet fever in man.

An analysis of the cases examined by Dr. Klein is shown in the following Table:

—	Name of patient.	Condition of tonsils.	Source of blood.	Micro-organisms isolated.	Death or recovery.
1	Lilian Fuller, aged 5	Severely ulcerated	Finger	Streptococcus	Ultimately recovered.
2	Kate Fuller, aged 2	Much ulcerated	„	Staphylococcus pyogenes aureus; Liquefying micrococcus; Streptococcus	Died of pyæmia.
3	Henry Lampson, aged 8	Ulcerated	„	Staphylococcus; Streptococcus	Recovered.
4	—— (a woman), aged 40	Much ulcerated	Arm	None	[Not stated.]
5	—— (a girl), aged 19	„	„	„	„
6	Blanche Moody, aged 15	Ulcerated	Finger	Streptococcus	Recovered.
7	Ellen Warr, aged 22	Much ulcerated	„	None	[Not stated.]
8	Robert Hughes, aged 8	Ulcerated	„	„	„
9	Florence Glossop, aged 2½	„	Heart	Streptococcus	Died.
10	Edward Fenton, aged 3	—	„	None	„
11	R. Boucher, aged 20 months	Advanced ulceration	„	„	„

The occurrence of a streptococcus in certain cases of scarlet fever had been observed by several previous investigators—Crooke, Löffler, Babes, Heubner and Bahrdt, and notably by Fränkel and Freudenberg.

Crooke, in cases of scarlet fever with severely affected throat, found bacilli, cocci, and streptococci in the organs of the throat, and cocci in the internal organs. Crooke left it an open question whether those cocci were the specific organisms of scarlet fever, or to be regarded as diphtheritic or septic associates. He inclined on clinical grounds to the latter view.

Löffler, in cases of scarlatinal diphtheria, found the same chain-forming micrococcus which he had found in typical diphtheria.

Babes was able to constantly prove in inflammatory products secondary to scarlatina the presence of a streptococcus greatly resembling that in pus.

Heubner and Bahrdt, in a fatal case of scarlet fever in a boy, complicated with suppuration of the finger- and knee-joints and

with pericarditis, found a streptococcus identical in form, from his description, with *Streptococcus pyogenes*. Cultivations were not made. The secondary infection started from diphtheritically affected tonsils, which were followed by retro-pharyngeal abscesses.

Fränkel and Freudenberg examined for micro-organisms three cases of scarlatina with well-marked affection of the throat. In all three cases they obtained cultivations of cocci from the submaxillary lymphatic glands, spleen, liver, and kidney. These cocci could in no way be distinguished from *Streptococcus pyogenes* derived from pus, nor from the undoubtedly identical streptococcus which one of them (A. Fränkel) had already repeatedly cultivated in large numbers from puerperal affections. In two of the cases *Streptococcus pyogenes* was the only organism present, and in all three cases it was far in excess of other colonies which developed. The organisms were also found in sections of the organs by microscopical examination. The identity of this streptococcus with *Streptococcus pyogenes* and *Streptococcus puerperalis* was established by comparison of their macroscopical and microscopical appearances in cultivations on nutrient agar-agar, nutrient gelatine, and in broth, both at the ordinary and at higher temperatures, and also by experiments on animals. They concluded that it could be stated with certainty that the organisms in question did *not* stand in causal relation to scarlet fever. They considered that special methods of microscopical and biological research were apparently needed for demonstrating the true scarlet fever contagium, which probably was especially present in the skin. They considered that the presence of the streptococcus was due to a secondary infection, to which the door was opened by the lesions of the throat—a view which was supported by the fact that the organisms were found in the submaxillary lymphatic glands. They preferred to use the term "secondary" to "complicated" or "combined" infection, because this expresses the fact that by the effect of the scarlatinal virus the soil is rendered suitable for this ubiquitous microbe when it has once gained an entrance.

Fränkel and Freudenberg could in no way distinguish the streptococcus in scarlatina from the streptococcus in pyæmia and septicæmia. I will therefore proceed to give a short account of the bacteriological researches in pyæmia and septicæmia.

Rosenbach examined six cases of pyæmia. From Case 1 cultivations of *Streptococcus pyogenes* were obtained from the blood of

the patient during life. The blood was stroked over the surface of the culture medium, and in two tubes the *Staphylococcus pyogenes aureus* was also present. In Case 2, with suppurative pleuritis, the pleura was tapped during life, and cultivations of *Streptococcus pyogenes* were obtained. In Case 3, streptococci were found in the metastases in the kidneys and in the other suppurations that were examined. In Case 4, the pleura was opened during life, and *Streptococcus pyogenes* and *Staphylococcus pyogenes aureus* were isolated from the fluid which escaped. In Case 5, pure cultivations of *Streptococcus pyogenes* were obtained from pus from the knee, which was punctured during life. This case was one of erysipelas and pyæmia after removal of a carcinoma of the breast. In Case 6, a case of whitlow with metastatic abscesses, the patient recovered, and no streptococci were found. Thus, in six cases of metastatic pyæmia *Streptococcus pyogenes* was found five times, partly in the blood and partly in the metastatic deposits, *and twice in company with Staphylococcus pyogenes aureus.*

Baumgarten also found the *Streptococcus pyogenes* in the internal organs in pyæmic cases, and Eiselsberg found *Streptococcus pyogenes in company with Staphylococcus pyogenes aureus in the blood of cases of septicæmia.*

In Case 2 (Kate Fuller, who died of pyæmia), it will be observed that Dr. Klein also found the streptococcus in association with *Staphylococcus pyogenes aureus.* Yet Dr. Klein maintains that the streptococcus found in scarlatina, in pyæmia and septicæmia, puerperal fever and suppuration, are different organisms, which can be readily distinguished by their different pathogenic properties. In a paper read before the Epidemiological Society Dr. Klein has published the data on which he has based this opinion. An examination of the following table shows that in no case are parallel experiments quoted:

	Streptococcus from puerperal septicæmia.	Streptococcus from pus.	Streptococcus from scarlet fever.
Rabbits	Local rash	Local suppuration	[Not stated.]
White mice	Fatal	„	Fatal in (small proportion).
Brown mice	[Not stated]	„	Fatal (in large proportion).

I must observe that the data which are here *quoted* by Dr. Klein have not been confirmed by a number of independent investigators. I refer to the pathogenic properties of the streptococcus when isolated from pus. The production of local suppuration does not follow inoculation of rabbits with *Streptococcus pyogenes*. This has been especially worked out by a number of independent investigators in comparing the action of the streptococcus from erysipelas with that from pus. When these micro-organisms were first described a great difference was believed to exist in their effect on animals, but numerous experiments on rabbits have distinctly proved that a local rash is equally the result of inoculating *Streptococcus pyogenes*. The streptococcus from scarlet fever, from pus, and from puerperal septicæmia may be described from the researches of Passet, Biondi, Eiselsberg, Baumgarten, Fränkel, Freudenberg, Flügge, and others, as producing local rash in rabbits, and as fatal in white mice in a small and in brown mice in a large proportion of the animals experimented on.

I cannot agree therefore with the conclusions drawn by Dr. Klein, as in the light of the researches made up to the present time, the statement that these organisms especially differ in their effects on animals is shown to be based on inaccurate data.

I have mentioned that Fränkel isolated a streptococcus from puerperal fever, which he at first called *Streptococcus puerperalis*, but subsequently identified with *Streptococcus pyogenes*. These researches have been confirmed by others. Winkel obtained a pure cultivation of a streptococcus from the blood of the heart in a case of puerperal peritonitis. It produced erysipelatous redness when inoculated in the rabbit's ear, and in form and in cultivation was similar to the streptococcus in erysipelas. Cushing also found *Streptococcus pyogenes* associated with puerperal infection. The cocci were found in endometritis diphtheritica as well as in secondary puerperal inflammation. These observations were still further confirmed by Baumgarten, and Bumm isolated the same organism in puerperal mastitis.

Thus the researches of Fränkel and Freudenberg are confirmed by a number of independent investigators, and, moreover, so far from being invalidated by the later researches of Dr. Klein, they are, on the contrary, by a correct interpretation confirmed by them. For I have already shown that the visceral disease produced in the calves inoculated by Dr. Klein was due to septicæmia, and there is

no evidence to show that the calves suffered from scarlatina complicated with septicæmia.

Therefore, to maintain that this streptococcus is the true cause of scarlet fever is not in accordance with the accepted teachings of bacteriology. The streptococci were only found in certain cases, and when cultivated artificially and inoculated *en masse* they did not produce scarlatina. This assertion is not even in accordance with the teachings which have been adopted and thus expressed by Dr. Klein himself, 'Micro-organisms and Disease':

"In all investigations of the relation of micro-organisms to disease, it is necessary to bear in mind that, as Koch has pointed out, no observation can be said to be complete, or one should rather say, in no instance can it be said to have been *satisfactorily proved*, that a particular infectious disease is due to a particular micro-organism, if any one of the following conditions remain unfulfilled."

Among these conditions we find the following:

"After having thus cultivated the micro-organism for several successive generations, it is necessary to reintroduce them into the body of a healthy animal susceptible to the disease, and in this way to show that this animal becomes affected with the same disease as the one from which the organisms were originally derived."

That this condition was not complied with is acknowledged by Dr. Klein, who states that "If proof is expected that fever, ulceration of the tonsils, and scarlatinal rash should be produced in the calf by the virus of scarlet fever, I am afraid such proof will not be forthcoming."

My own opinion as to the relation of this streptococcus, when found in scarlatina, to the disease itself, coincides with the conclusions of Fränkel and Freudenberg. For some time my attention has been drawn to the probable identity of the streptococci in suppuration, puerperal septicæmia, pyæmia, and septicæmia, and in certain cases of measles, scarlatina, and diphtheria. This I expressed early in 1886. Thus, in describing the streptococcus *in* puerperal fever, I wrote as follows:

"In this and many similar cases the cocci are given as distinct species from their association with particular diseases, not because they are believed to be causally related, for there is very little evidence in favour of that belief as yet, but purely for convenience of reference. In many cases they are only septic organisms which

have found a pabulum in the dead tissue; others appear to be identical with organisms which have been found in pus."

In the following year, May, 1887, I received from Dr. Klein himself a cultivation on nutrient gelatine and a microscopical preparation of his streptococcus. The appearances (Pl. XXX) obviously corresponded with well-known streptococci. But if it were a fact that this streptococcus actually produced scarlet fever, it would be entitled to be regarded, in opposition to the conclusions of Fränkel and Freudenberg, if not as a distinct species, at least a variety with characteristic pathogenic properties. Since that date the details of Dr. Klein's experiments with this streptococcus have been published, and there are no grounds for believing from these experiments that this streptococcus differs in its pathogenic properties from the streptococcus associated with septic infection.

Further, I have been able to confirm my opinion, and the researches of the bacteriologists in Germany, who have insisted upon the fact that the streptococcus in pyæmia and the streptococcus in scarlet fever are one and the same micro-organism. The following case will serve to illustrate this. An out-patient with chronic abscesses and pyæmia was admitted into King's College Hospital. Sir Joseph Lister punctured two abscesses in the sacral region and the shoulder, and the pus from both, amounting to about 12 oz., was collected with all the necessary precautions, and handed over to me for examination. I found that the pus contained a pure cultivation of this streptococcus. Now, Dr. Klein has stated that the streptococcus which he has isolated from certain cases of scarlet fever is *morphologically distinguishable*[1] from the streptococcus from any other source. I cannot agree with this statement, and as cases of scarlet fever and pyæmia are often within reach of pathologists in our large hospitals, I will take this opportunity of describing some of the practical details of this part of my investigation which will enable those who are in a position to examine such cases to repeat the experiments, and thus convince themselves that Dr. Klein's statement is quite erroneous. First of all, the pus may be examined microscopically; to do this I make use of an ordinary platinum inoculating needle, which is bent at the extremity into a hooklet; this is sterilised and dipped into the pus, which is then

[1] See page 376 of the Supplement to the 'Sixteenth Annual Report Local Government Board, 1886—1887,' containing the Report of the Medical Officer for 1886.

DESCRIPTION OF PLATE XXX.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

From a microscopical specimen of the so-called scarlet-fever streptococcus. This specimen, dated May 5th, 1887, was prepared by Dr. Klein, and a drawing is given so that a comparison may be made with the drawing of the streptococcus from pyæmia under precisely the same conditions of amplification and illumination. × 1200. Powell and Lealand's apochromatic $\frac{1}{12}$ Hom. imm. E. P. 10.

STREPTOCOCCUS FROM CERTAIN CASES OF SCARLET FEVER X 1200.

From Specimen prepared by Dr Klein May 5th 1887

Drawn by Leonhard Apochromatic 1/12 Hom Imm E P 10.

Brookshank del.

Fred[k] Dangerfield Lith. London 16423.

DESCRIPTION OF PLATE XXXI.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

From a microscopical specimen prepared from pus from a pyæmic abscess. Stained with gentian-violet by the method of Gram, and contrast-stained with eosin. × 1200. Powell and Lealand's apochromatic $\frac{1}{12}$ Hom. imm. E. P. 10.

STREPTOCOCCUS FROM PYÆMIA. X 1200.

Powell & Lealand Apochromatic ⅛ Hom imm. E P 10

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DESCRIPTION OF PLATE XXXII.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From drawings by Mrs. Crookshank.

Artificial cultivations of streptococcus from pyæmia.

FIG. 1.—On the surface of nutrient gelatine.
FIG. 2.—On the surface of nutrient agar-agar.
FIG. 3.—In the depth of nutrient gelatine.

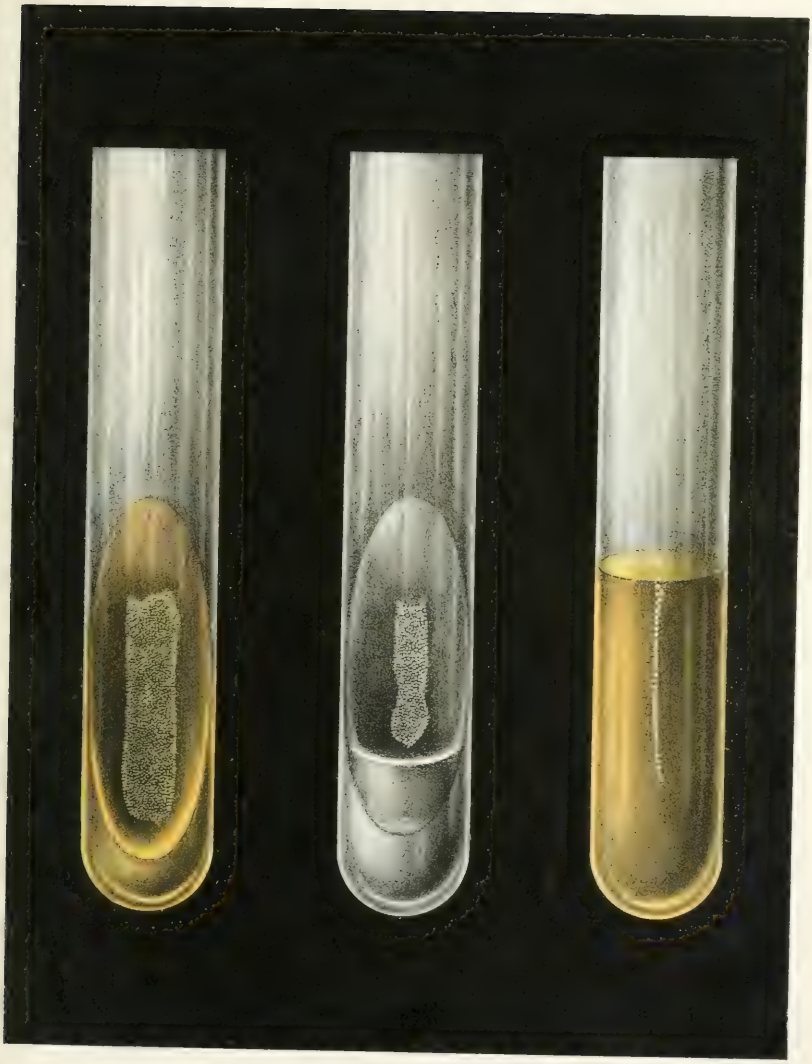


Fig. 1. Fig. 2. Fig. 3.

H. Crookshank del.

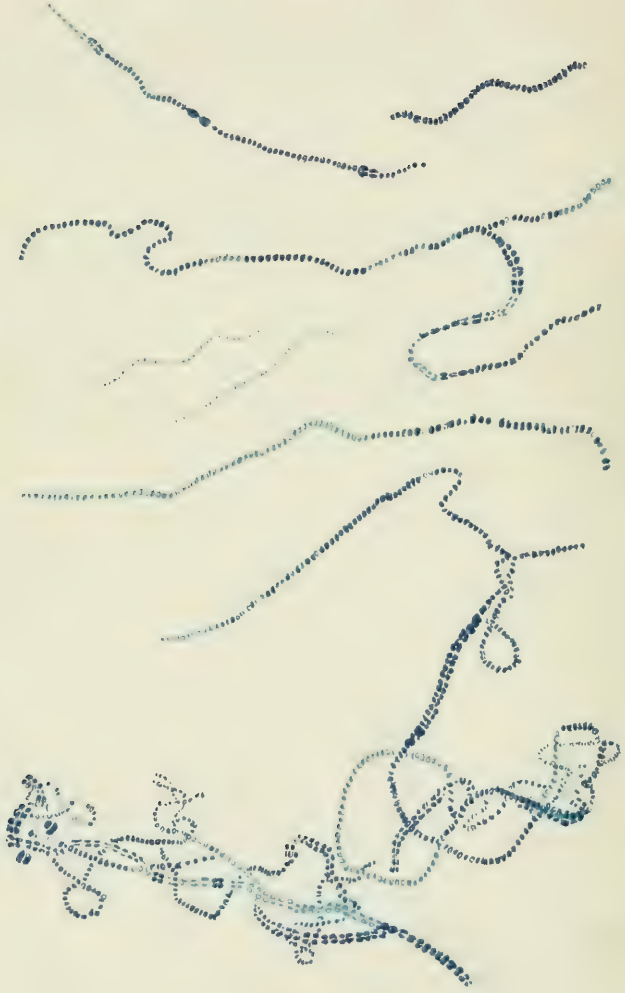
Mintern Bros. imp.

spread out on a cover-glass into as thin a film as possible; the preparation is treated in the ordinary way, that is to say, the film is allowed to dry, and the cover is taken up with forceps, and passed three times through the flame with its prepared side uppermost. To stain the preparation I employ the method of Gram and eosin. In this way the streptococci are stained blue, and stand out in marked contrast to the rest of the preparation. I prefer to use freshly prepared solution, which is made as follows :— Place four or five drops of pure aniline oil in a test-tube, fill it three quarters full with distilled water, close the mouth of the tube with the thumb, and shake it up thoroughly; filter the emulsion twice, and pour the filtrate into a watch-glass or glass capsule; to the perfectly clear aniline water thus obtained add drop by drop a concentrated alcoholic solution of gentian violet until precipitation commences. The method of staining is as follows:—Float the cover-glasses on the solution for ten minutes to half an hour, then transfer them to iodine-potassic-iodide solution, until they assume the colour of a tea leaf; then immerse them in alcohol until they are decolourised; dip them in an alcoholic solution of eosin for a few moments, and then transfer them to clove oil to clarify the film; to remove the clove oil gently press the cover between two layers of clean filter paper, then mount in xylol-balsam. (Plate XXXI.)

The best method for cultivating the organism is to employ a similarly shaped platinum wire, and to spread a droplet of the pus or blood of the patient over the surface of nutrient agar-agar solidified obliquely. The tubes are then placed in the incubator at 37° C.; the streptococcus will appear in the course of two or three days in the form of minute dotted colonies. (Plate XXXII.) If present alone, and in considerable quantities, the inoculated surface will exhibit a pure cultivation consisting of a number of such colonies, whilst a flocculent mass is observed in the liquid which collects at the bottom of the agar-agar tubes; this flocculent mass will be found to be composed of chains. From such a tube I inoculate a number of the small flasks employed in M. Pasteur's laboratory for cultivations in liquids. In this way a number of pure cultivations in milk and broth are established, which can be readily examined from time to time. From a pure cultivation in broth or agar-agar, tubes of nutrient gelatine can be inoculated. (Plate XXXII.) Cover-preparations from the growths on solid media can be made in

the usual way, and stained with either a watery solution of fuchsin or gentian-violet; but to stain preparations made from milk or broth, or from the liquid in agar-agar tubes, I prefer to use the method of Gram, the stain will then be removed except from the streptococci, and very beautiful preparations result. (Plate XXXIII.)

Such preparations should be studied minutely with the best lenses and the best method of illumination in order to make out all the minute details. The individual elements composing the chains will be found to vary considerably in size; here and there in a preparation will be found a chain composed of excessively small elements, in another part the elements are all on a larger scale, and again in another part the elements will be peculiarly conspicuous on account of their size. Very characteristic appearances result from the fact that the elements enlarge and divide both longitudinally and transversely, and, indeed, the largest elements, for the most part, clearly show a division in two directions, resulting in the formation of tetrads. So great is the diversity in the size of the elements of some of the chains that one might imagine that there was more than one kind of streptococcus present in a preparation until on examining some of the longest chains it is observed that various sizes are represented in different lengths of the same chain. In addition to the forms resulting from the fission of the elements, there are here and there in a chain, and sometimes terminally, larger elements, which are spherical, spindle-shaped, or in the form of a lemon. In the length of the chains, as in the size of the individual elements, there is usually a great diversity. In some cases they are composed of only a few, three or four elements, or in others eight, ten, or twenty. Here and there an exquisite rosary will extend in a straight line across the field of the microscope, or be twisted, curved, or serpentine; in some preparations twisted or entangled strands are observed which are composed of several hundred elements. Such chains will be found to be much thicker in one part than another. Another characteristic appearance is produced by separation of the elements resulting from fission in the long direction of the chain, by which lateral twigs or branches are formed. Another character, which is very striking, may be seen when the individuals in a chain have become separated, an unstained or faintly stained membrane may be found bridging across the interval. This will become still more visible in some of the preparations contrast-stained with eosin. I



STREPTOCOCCUS FROM PYÆMIA.

Powell & Leland Apochromatic 1/12 Hom. Imm. E. P. 10.

E. Crookshank del.

Fred^k Dangerfield Lith. London. 16423

DESCRIPTION OF PLATE XXXIII.

To illustrate the paper of Prof. Crookshank on the so-called Hendon Cow Disease in its Relation to Scarlet Fever in Man. (Page 371.)

From a drawing by himself.

From microscopical specimens of artificial cultivations of the streptococcus in broth and in milk at different stages of growth.

In these preparations there is a great diversity in size and form of the chains and their component elements. In the drawing examples are figured of the following:

a. Chains in which the elements are more or less uniform in size.

b. Chains with spherical and spindle-shaped elements at irregular intervals. These are conspicuous by their size and are sometimes terminal.

c. Simple chains composed of elements much smaller than the average size.

d. Branched chains.

e. Complex chains with elements dividing both longitudinally and transversely, and varying considerably in size in different lengths of the same chain. × 1200. Powell and Lealand's apochromatic $\frac{1}{12}$ Hom. imm. E. P. 10.

am inclined to regard this as a sheath analogous to the sheath which, in the leptothrix filaments of anthrax, surrounds the individual rods of which the filament is composed. [Plates XXXI and XXXIII.]

The evidence I have brought forward is sufficient to show that in any disease accompanied with a lesion of the skin or mucous membrane, in which the blood and tissues are profoundly affected by the virus of that disease, micro-organisms gain an entrance into the circulation, and may not only escape destruction but may gain the upper hand and set up destructive processes. And it is interesting to observe in connection with this, that the streptococcus was isolated in all cases at or about the time at which the temperature was highest; in other words, at a time when the tissue resistance to an invasion of micro-organisms would be lowered. Occurrence, therefore, of this streptococcus, which has been designated *Streptococcus pyogenes*, in certain cases of diphtheria, scarlet fever, vaccinia, variola, measles, typhoid fever, must be regarded as a secondary result associated with suppuration or with septic or pyæmic complication; and thus I am forced to the conclusion that, excepting negative evidence, bacteriology has not assisted us the least in the determination of the real nature of the morbific agent or actual contagium of the exanthemata. That there are certain diseases, such as anthrax, chicken cholera, tuberculosis, septicæmia of mice, in which a micro-organism is the principal agent in producing the disease, is, I am sure, accepted by all or nearly all modern pathologists; but we are nevertheless far from having solved the nature of the contagium of all communicable diseases. In many cases, as in hydrophobia, where bacteria have been from time to time brought forward as the specific agents, they have been gradually substituted by the less definable term "virus." Bacteriology misapplied in pathology will be both misleading and mischievous, and I maintain that this criticism is applicable if a micro-organism is regarded without doubt as the actual contagium of a disease, such as scarlatina in man, in the face of obvious fallacies, and without its affording the general and experimental evidence which is usually regarded as necessary for admitting a causal connection between micro-organisms and disease.

As bearing upon this question of the etiology of scarlet fever, I would observe that Dr. Klein regarded it as an important piece of evidence that in a certain brand of condensed milk under suspicion

of producing scarlatina, a streptococcus was found which had a "striking resemblance" in its morphological and cultural characters to the streptococcus of scarlatina, and further, in a monkey supposed to have died of scarlet fever, a similar micro-organism was found. This monkey died in a house where scarlet fever had occurred. From the presence of this streptococcus and from the anatomical condition of the viscera, Dr. Klein regarded as without doubt the unity of this unknown disease with acute scarlatina in man. The post-mortem appearances, however, were obviously from the description the post-mortem appearances of septicæmia.

Formerly certain differences in the result of inoculation of animals were regarded as establishing a distinction of species; but further researches have shown that often these differences are not constant, and, moreover, in the history of the well-known pathogenic organisms we meet with different effects produced by one and the same species. Indeed, we find that the pathogenic effect depends upon several conditions, such as the source of the micro-organism, the dose, the mode and place of infection, and the nature of the inoculated soil. The principal method of attenuation of virus depends upon the fact that by passing organisms through different animals they assume different properties. The anthrax bacilli from sheep are fatal to sheep, but if passed through mice produce a transitory illness. The microbe of swine erysipelas passed through pigeons is increased in pathogenic power, but by passing through rabbits is progressively diminished, producing only a mild disease. But not only is the pathogenic effect different, but the organism itself may be altered. Thus, according to Huber, anthrax bacilli from the guinea-pig are thicker than when they are derived from the mouse or sheep, and the latter are thicker than in the rabbit.

The bacilli of tuberculosis of birds, of cattle, and of man differ slightly in their size and their distribution in the tissues. Further, corresponding with the slightest deviation from the typical form of a micro-organism there will be correspondingly minute differences in the appearances to the naked eye of the cultivations; so that we must not be too ready to deny the identity of two micro-organisms which only differ in some excessively minute difference in appearance, in form, in cultures, or in effect. Therefore, because there was not a complete identity but only a striking resemblance in the appearances on cultivation and under the microscope, we must not

on this ground be too ready to exclude the evidence of the condensed milk.

What really renders this evidence of practically no value whatever is the fact that in a vast number of diseases and conditions there are found streptococci which, in their characters on cultivations, and under the microscope, and in their pathogenic properties, are not to be distinguished with certainty or at all from the streptococcus in scarlatina.

I will proceed to enumerate these conditions.

Diseases and conditions in which streptococci are found which have been proved to be in some cases identical, in others strikingly similar to the streptococcus occasionally found in scarlatina in man.

I have already described the occurrence of a streptococcus in pyæmia and septicæmia and puerperal septicæmia. In abscesses a chain-forming micrococcus was first described by Ogston. It was later studied by the methods of cultivation introduced by Koch and named by Rosenbach *Streptococcus pyogenes.* According to Flügge, after subcutaneous inoculation of mice with a small quantity of a cultivation, there is no result in 80 per cent. of the animals experimented on. Sometimes there is limited pus-formation at the seat of inoculation, sometimes the animals die without any very striking pathological appearances.

In three cases of typical diphtheria Löffler found a streptococcus. He isolated it by cultivation, found that it was similar in form, characters on cultivation, and effects after inoculation, to Fehleisen's streptococcus of erysipelas. Löffler was not inclined to regard them as identical, because Fehleisen never found his cocci in the blood-vessels. Flügge named the organism *Streptococcus articulorum,* and states that, after subcutaneous inoculation or injection of a cultivation in mice, a large proportion of the animals die, and in the sections of the spleen and other organs the streptococci are again seen. Baumgarten investigated the same subject, and decided that the streptococcus was identical with *Streptococcus pyogenes.*

In erysipelas Fehleisen isolated a streptococcus and described the appearances on cultivation. Fehleisen regarded the organism as quite distinctive, with special characters on cultivation; but

Rosenbach pointed out the extremely close resemblance in every respect to the *Streptococcus pyogenes*, though he described an apparent difference on cultivation on nutrient agar-agar. Further investigation, however, by other observers showed that these organisms could not be distinguished with certainty by either their morphological appearances or by their characters on cultivation. Moreover, the marked differences which have been described after inoculation were not obtained by those who repeated the experiments. Passet found that the results of inoculating *Streptococcus pyogenes* in the rabbit's ear induced a very similar condition to the result of inoculation of *Streptococcus erysipelatis;* and both organisms, by subcutaneous inoculation, and by inoculation in the cornea of rabbits and other animals, induced results without any constant difference. Passet showed that the inoculation of the cornea produced the same form of keratitis. Hoffa and Hajek described minute differences at the seat of inoculation, but Biondi and Eiselsberg repeated the experiments, and failed to establish the alleged differences. Baumgarten also investigated this subject, and failed to prove any essential difference, and, indeed, found much more often than might have been expected from the publications of previous authors that no marked result at all was obtained on inoculation; and he concluded that *Streptococcus pyogenes* and *Streptococcus erysipelatis* in their form, cultivation, and their effects on animals were identical. Thus Passet, Biondi, Eiselsberg, Baumgarten, and Fränkel have definitely accepted the identity of the streptococcus associated with suppuration, and the streptococcus associated with erysipelas.

From a case of spreading gangrene, which was identical with Ogston's erysipelatoid wound gangrene, and regarded by him as the most intense and dangerous form of erysipelas, Rosenbach obtained pure cultivations of a streptococcus by incising the skin of the limb, and inoculating tubes from the turbid reddish fluid which escaped. That the streptococcus was identical with *Streptococcus pyogenes* was ascertained by comparison, with a cultivation derived from pus, of the mode of growth, and of the effect on animals.

Surgical fever.—Eiselsberg proved the presence of a streptococcus in the blood of several cases of surgical fever in Billroth's clinic. The organism was identified by cultivation with *Streptococcus pyogenes.*

In vaccine lymph, Cohn and others have recognised the presence of micro-organisms. Guttman and Pfeiffer have separated the different species by Koch's methods. Guttman found —(1) *Micrococcus pyogenes aureus;* (2) *Staphylococcus viridis flavescens;* (3) *Staphylococcus cereus albus.* (Syn. *Micrococcus vacciniæ*, Cohn, Voigt, Garré). Dr. Klein has isolated a micrococcus which he calls *Streptococcus vacciniæ*, and states to be identical with one of Guttman's cocci. But the *Streptococcus pyogenes* may occur in vaccine lymph, and Pfeiffer regards it as the result of contamination. Hence, it is suggested that before calf lymph is employed for vaccination it should be tested on a rabbit's ear. If in two days no rash has been produced, the possibility of the presence in the lymph of *Streptococcus pyogenes* or *erysipelatis* is excluded. The nature of the contagium in vaccinia as in scarlet fever is unknown.

Hlava has established the presence of *Streptococcus pyogenes* in the pustules of variola, and Garré found streptococci in the internal organs in a case of variola hæmorrhagica.

In a fatal case of variola complicated with pemphigus, Garré found a streptococcus in the pemphigus vesicles. Whether it was identical with *Streptococcus erysipelatis* Garré left an open question.

In 1868 Professor Brown figured a streptococcus in the milk of cows affected with foot-and-mouth disease. From the vesicles of this disease in sheep, Dr. Klein isolated a streptococcus which he regarded as the contagium of the disease. From Dr. Klein's description of its morphological features and characters on cultivation it closely corresponds with *Streptococcus pyogenes.* Mr. Watson Cheyne investigated the same disease, and informs me that he also isolated a streptococcus, which he concluded was *Streptococcus pyogenes.* Baumgarten also regards Dr. Klein's alleged contagium as most probably the *Streptococcus pyogenes.* Against the fact of this organism being the virus of this disease, and in favour of its being *Streptococcus pyogenes*, we have the results of inoculation experiments. A great number of subcutaneous inoculations in sheep were without any result. One of several guinea-pigs fed with the organism had an abscess, another an ulcer. In sheep fed with the organism, two out of nine, after a number of repeated administrations, developed a disease, regarded by Dr. Klein as foot-and-mouth disease. There is the possibility of two fallacies—

one that the disease, if really foot-and-mouth disease, may have occurred incidentally; the other, that the result which occurred may have been a spurious form of foot-and-mouth disease. The control experiment of exposing a number of sheep with these two sheep to see if the disease would spread was wanting.

Semner cultivated streptococci from the blood and lymphatic glands of a sheep suffering from cattle plague. A calf inoculated with a cultivation was stated to have died in seven days from cattle plague. The cocci were stated to lose their virulence by successive cultivation, and the weakened cultivation to protect against the virulent disease. I think it extremely likely this was another manifestation of *Streptococcus pyogenes*, and that the animal died very probably of septic infection. Dr. Klein is also of opinion, in spite of Semner's statements, that the specific nature of these micro-organisms cannot be considered to be established.

In a severe case of swine fever, with well-marked intestinal ulceration, I have obtained from the blood of the spleen a mixed cultivation of streptococci and other organisms. Isolation and cultivation in the pure state are not yet completed. It was very probably mixed infection with *Streptococcus pyogenes*.

Babes observed the presence of streptococci in the vessels of the kidney and liver in yellow fever. Cultivation experiments are wanting. It was probably secondary infection with *Streptococcus pyogenes*.

Babes, in a case of fièvre bilieuse typhoide, found masses of streptococci filling the vessels of the liver, kidney, and spleen. Probably another instance of secondary infection with *Streptococcus pyogenes*.

From the blood and from inflammatory post-products in measles Babes isolated a streptococcus, which he describes as closely resembling the *Streptococcus pyogenes*.

Wyssokowitsch found cocci in the internal organs in ulcerative endocarditis, and produced the disease in animals after injury to the valves by injection of *Streptococcus pyogenes* and other organisms. Wechselbaum, by microscopical research and by cultivation experiments, proved the presence of *Streptococcus pyogenes* in acute verrucous endocarditis. Baumgarten confirmed this. He found *Streptococcus pyogenes* alone in one case and accompanied by *Staphylococcus aureus* in another.

Senger found a streptococcus in a case of typhoid with secondary

infection. This was probably *Streptococcus pyogenes*. Dunin found the well-known pyogenic organisms in post-typhoid suppuration, mostly as staphylococci, but sometimes streptococci.

Wechselbaum found the *Streptococcus pneumoniæ*, which resembled *Streptococcus pyogenes* and *erysipelatis* morphologically and on cultivation. It was found in twenty-one cases of pneumonia by microscopical research, and cultivated in nineteen. It had no effect on the rabbit's ear. Baumgarten, nevertheless, regards this as *Streptococcus pyogenes*, and is of opinion that it is only a matter of further research to establish that view.

Neumann found streptococci by microscopical research in the lungs, and by cultivation isolated a streptococcus in pneumonia after typhoid. It corresponds with Wechselbaum's streptococcus in not affecting the rabbit's ear.

Rosenbach obtained a pure cultivation of *Streptococcus pyogenes* in a case of empyema. Wechselbaum in two cases also established its presence.

Thaon found a streptococcus in the lungs of children in fatal cases of broncho-pneumonia complicating measles, diphtheria, and whooping-cough. It was regarded as identical with the streptococcus isolated by Löffler from diphtheria. Fränkel discovered a streptococcus in the lungs of a case of true croup complicated with broncho-pneumonia, and by cultivation established its identity with *Streptococcus pyogenes*.

Koch produced a disease in mice by subcutaneous injection of putrid blood. In tissue sections a chain coccus was found, and Baumgarten is of opinion that it is very probably identical with the *Streptococcus pyogenes;* but cultivations are still wanting.

Charrin found cocci in rabbits, examined some hours after death, from anthrax. These, when isolated, produced death in rabbits from septicæmia without suppuration. Chains composed of from fifteen to twenty elements were found in all the organs. This was probably another instance of *Streptococcus pyogenes*.

Kassowitz and Hochsinger found the presence of a streptococcus in the tissues and internal organs, and especially in the blood-vessels, in fatal cases of congenital syphilis. These observers regarded their discovery as having an important bearing on the etiology of syphilis, but Kolisko pointed out that it was only the result of septic infection with presence of *Streptococcus pyogenes*, as had already been established in scarlet fever.

From the meningeal exudation of a case of apparently idiopathic cerebro-meningitis Banti found by Koch's methods the presence of *Streptococcus pyogenes* and *Staphylococcus aureus* and *albus*. The cocci probably entered through an abscess of the jejunum.

Widmark isolated by cultivations *Streptococcus pyogenes* and other organisms from cases of blepharadenitis and phlegmonous dacryocystis. In plegmonous dacryocystis Widmark found *Streptococcus pyogenes* almost exclusively.

Flügge cultivated a streptococcus from necrotic patches in the spleen of a fatal case of leukæmia. Cultures corresponded very closely with *Streptococcus pyogenes*. Inoculation in the ears of rabbits produced similar results to *Streptococcus pyogenes* or *erysipelatis*. Flügge calls it *Streptococcus pyogenes malignus*, but concludes that it is probably identical with the streptococcus from pus.

Nicolaier, and later Guarneri, isolated a streptococcus from soil. Microscopically it could not be distinguished from other streptococci. Baumgarten is of opinion that it is neither in form nor in cultivation to be distinguished with certainty from *Streptococcus pyogenes*.

Emmerich succeeded in proving the presence of streptococci in the air of a hospital where erysipelas had broken out. These cocci in their form, their characters on cultivation, and in inoculation results, were identified with the *Streptococcus erysipelatis*.

Baumgarten points out that in the most various substances undergoing putrefaction streptococci are always found which morphologically are exactly similar to the *Streptococcus erysipelatis*. Baumgarten, therefore, regards it as quite possible that the *Streptococcus erysipelatis* or *pyogenes* is essentially a saprophytic organism —that is to say, an organism which may be found wherever there is decaying organic matter—and that its parasitism is only incidental.

From the evidence I have adduced, and from the facts I have described, I am led to the belief that: 1. *The nature of the contagium of scarlet fever is unknown*. 2. The streptococcus regarded by Dr. Klein as the contagium is a septic microbe. 3. This streptococcus is found, sometimes in company with *Staphylococcus pyogenes aureus* as a secondary result in scarlet fever and many other diseases. 4. A streptococcus was first observed in scarlet fever by Crooke, later by Löffler, and by Heubner and Bahrdt;

but its exact relation to scarlet fever and its identity with the streptococcus from pus and puerperal fever were definitely established in 1885 by Fränkel and Freudenberg.

December 15*th*, 1887, *and January* 17*th*, 1888.

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2. *A note on the histology of sterile incubated cancerous and healthy tissues.*[1]

By Samuel G. Shattock and Charles A. Ballance.

[With Plates XXXIV and XXXV.]

In our previous communication we were able to show that portions of cancer (*i. e.* carcinoma or sarcoma) and portions of normal tissues, viz. muscle, kidney, testicle, liver, submaxillary salivary gland, could be maintained sterile upon blood-serum, agar-agar, and in nutrient jelly for an indefinite period at a temperature of 100° F. Some of the tubes which we had the honour of submitting to the Society this time last year we are able to show this evening; they are still sterile, and the cancer lumps within them appear as though only just removed from the operating room.

In these experiments no antiseptics were used; the precise details of the method are described in the former paper.

We now propose to record briefly the histological appearances exhibited by sterile incubated tissues, cancerous and healthy. We had made some observations on this subject at the time our paper was read last year, but we did not think that we should be justified in bringing them before the Society until we had made further observations, and the matter still needs, indeed, much more investigation. We have during the last year incubated portions of nearly forty malignant tumours, and have made a large number of experiments with healthy tissues. These have been submitted after incubation to microscopical examination. The staining reagents employed have been logwood and fuchsin, as we found that they

[1] Towards the expenses of this research a grant was made by the British Medical Association, on the recommendation of the Scientific Grants Committee of the Association.

best show the nuclear changes, and the microscopical examinations were made with $\frac{1}{2}\frac{1}{5}$ (Zeiss) water immersion, combined with Ocular 1 or 3; Reichert's $\frac{1}{1}\frac{1}{5}$ homogeneous immersion, Oc. 3; Zeiss's apochromatic $\frac{1}{1}\frac{1}{2}$ homogeneous immersion Oc. 8. The phenomena have been observed when the piece of cancer has been allowed to rest upon a solid medium above the drop of fluid which always collects at the lower end of the test-tube. If it soaks in the water, or if micro-organisms grow within the tube, or if the tube contain fluid nutrient jelly, the specimen becomes spoilt in a great measure for subsequent examination. The portion of cancer between its removal from the patient and its transference to the incubator must be maintained at a temperature of 100° F.

The appearances in question we have observed in both incubated and in unincubated cancer, and from numerous observations we can affirm that similar changes are not to be observed in normal tissues under similar circumstances.

These changes we may describe in detail from a portion of mammary scirrhus, after eight days' incubation, the specimen being transferred to Müller's fluid, and then to spirit, after having been in the tube for ninety days, and remaining sterile for the whole of this period. The tumour was removed on February 7th, 1887, from a widow, aged 51, whose mother had died of cancer of the breast.

The patient had had inflammation of both breasts during lactation.

Three years ago she noticed a small hard swelling at the outer border of the left breast; this gradually increased, and at the date of operation was of considerable size. The superjacent skin was adherent for about two and a half inches; tumour movable over the pectoralis. The axillary and cervical glands were diseased.

Epithelium.—The outline of the cell-protoplasm is not visible. The nuclei of the epithelial cells are very granular, the nuclear matrix between the granules being either slightly stained or colourless. In some of the nuclei there is a distinct coloured network (nucleoplasm). There are in addition distinct granules of larger size, or nucleoli. A delicate continuous outline exists to all the nuclei. Many of the nuclei have a spinous border due to the projection of the peripheral of the granules noticed, the limit of the nucleus remaining clearly defined. In other cells the granules have passed beyond the proper margin of the nucleus, and lie in the cell-protoplasm, an extremely delicate, thread-like process or stalk still connecting the granule in many instances with the rest

of the nucleus. The granules vary in size on leaving the cell-nuclei. They are mostly spheroidal, and in many instances oval, or comma-shaped, or with a more delicate filament or tail. The matrix in some of the nuclei is uncoloured, and with comparatively few darker points; in some of these nuclei one or two granules remain attached to the nucleus by a delicate process, the nucleus itself being scarcely distinguishable. The explanation of this last appearance may be that the granules have all passed out of the nucleus. Similar granules occur here and there free in the loculi.

Fibrous tissue.—The nuclei of the corpuscles are of two sizes. The smaller are more or less circular in outline, and deeply stained throughout, the nuclear matrix and nucleoplasm being in most instances indistinguishable from each other. Around the nucleus the cell-protoplasm is discernible as a narrow hazy zone. At the circumference of the nucleus are numerous projections of various sizes, some sessile, others stalked, and others free of the nucleus; these are deeply stained, and still lie in the cell-protoplasm. The larger nuclei are variously shapen, some regularly oval and of considerable size, with distinct granules and clear matrix, others narrow and of less regular form, and darkly and uniformly stained; and around these latter are particles like those first noticed. In the case of the connective-tissue corpuscles, the buds are for the most part larger and less regular in size than those described in connection with the nuclei of the epithelial cells. In the fibrous tissues around the corpuscles are minute granules, presumably arising in one or both of the above ways, and passing into the clefts of the tissue; the granules, of which the smallest are hardly visible, are fairly uniformly, and thickly distributed.

These appearances we have observed in the incubated pieces of several carcinomata (and in sarcoma) though not to the above degree.

In a **second** case, for example, in which a portion of scirrhus of the breast had been incubated for thirty-three days and left fifty-five days in the tube, remaining sterile throughout, the appearances observed in sections stained with logwood are as follows:

Epithelium.—Nuclei sharp, well-defined, extrusion of coloured particles, some pedunculated, others free within the granular cell-protoplasm (the outline of the cell-body being distinct), others free in the alveolus. Unstained epithelial cells occur in the groups,

in which cells no nucleus is discernible. The granules in the alveoli are often distinctly comma-shaped.

Fibrous tissue.—The corpuscles in a few spots show budding of granules from the nucleus; in the fibrous tissue in their vicinity there are in places a few free granules.

The patient from whom the preceding specimen was obtained was aged 35, married; no family history of cancer; eight children, the last being five weeks old. Twelve months ago a small hard lump was noticed at the upper and inner part of the left breast. The tumour has been growing rapidly since the last confinement. The skin was infiltrated and adherent to the new growth, adherent to pectoralis; nipple not retracted. The axillary glands were diseased.

In a **third** case the tumour was a small scirrhus from the right breast of a single woman aged 50. No family history of cancer. The growth had been noticed for fifteen months; many axillary glands were diseased. The piece of tumour was incubated for twelve days and examined after having been in the tube altogether twenty days.

Nuclei of epithelium spinous; extrusion of coloured particles into the cell-protoplasm, the limits of which are easily seen; there are a few granules free of the cells; some of the granules are distinctly comma-shaped.

The nuclei of some of the smaller connective-tissue cells show budding from their margins.

In a **fourth** the tumour, from a private patient aged about 60, was adherent to the skin, which was not ulcerated and excoriated. Axillary glands diseased.

A portion incubated for five days and kept in the tube altogether eight days, shows in fuchsin and in logwood preparations the following appearances:

Epithelium.—Nuclei lightly stained with darker points sharply defined; cell-protoplasm unstained, nebulous. From the margins of the nuclei there project granules of deeply stained substance, some attached by stalks. These granules become disconnected with the nucleus so as to be free in the cell-protoplasm. In some of the alveoli there are similar granules which appear to be free. In other places the nucleus is divided into larger irregular parts.

Fibrous tissue.—Nuclei of corpuscles very granular; budding

of coloured substance in places; a few granules free in the tissue.

Another piece of this tumour was incubated in nutrient gelatine for five days and was kept in the tube altogether sixty-three days before hardening for section. The appearances presented by this preparation are the same as those just described.

In a **fifth** case the tumour was noticed nine months previously as a hard lump in the right breast the size of a walnut; has grown rapidly during the last four months. The patient was aged 52; twelve children, three miscarriages. The breast, on admission, was infiltrated with new growth, and about the size of an orange, not adherent to pectoralis or to skin; axillary glands extensively diseased.

A piece of the tumour incubated for seven days on agar, and placed after that period in Müller's fluid, &c., shows:

Epithelium.—Cell-protoplasm with difficulty discernible in most places, nuclei very granular; the nuclei present minute sessile eminences, and here and there a stalked or capitate process of deeply stained substance, in all respects like that forming the coloured portions of the nucleus. Free granules of similar kind occur in the alveoli and in the fibrous tissue.

In a **sixth** case, a small atrophic scirrhus of the breast from a woman aged 61, the tumour had been noticed for six months; there was a history of injury. The growth was nearly central, and had led to puckering of the superjacent skin. No glands were felt enlarged and none were removed at the operation. The following appearances are observable in sections of portion of the tumour after nine days' incubation:

Epithelium.—Nuclei very granular; budding of the nucleoplasm or chromatin from the margins of several of the nuclei; some of the extruded granules have fine stalks of considerable length, three or four times the diameter of the granule. Similar granules occur in the cell-protoplasm disconnected with the nucleus, and others free in the alveoli, and in the adjacent fibrous tissue.

Fibrous tissue.—Nuclei of corpuscles very granular; here and there fine budding occurs.

In a **seventh** case the tumour was from a single woman aged 41, and was a small atrophic scirrhus of the breast, situated to the inner side of the nipple. There were two hard glands felt in the axilla.

A piece of the tumour after eight days' incubation on agar, shows, in sections stained with fuchsin, a very granular condition of the epithelial nuclei, the margins of which show in places distinct bud-like extrusions of the chromatin; free granules occur amongst the epithelial masses, and in the adjoining fibrous tissue. The nuclei of the fibrous tissue present in a few spots similar appearances.

In an **eighth** case the patient was a nurse, aged 64, married, two children. Two years previously she had received a severe blow on the right mamma, and three months after this a small tumour appeared. No family history of cancer. The skin was infiltrated; tumour not adherent to pectoralis. Many of the axillary glands were diseased.

A piece of the tumour after incubation for eight days presents (in fuchsin preparations) similar budding from the epithelial nuclei, and in a few spots disconnected chromatic granules in the cell-protoplasm.

The nuclei of the connective-tissue corpuscles show distinct budding of a similar kind.

How far exactly the process of incubation aids in producing the appearances under notice we are not quite prepared to say. That the presence of free chromatic granules in carcinoma of the breast is not limited to the incubated specimens we can assert from observation.

As an example of an ordinary mammary carcinoma we may describe a portion of the tumour previously noticed under the fifth case, the material having been prepared quite fresh, in Müller's fluid and then in spirit, and the sections stained with fuchsin, and examined under a high power of the microscope.

Epithelium.—Cell-protoplasm lightly stained; nuclei oval; intra-nuclear network faintly stained or not distinguishable; nucleoli single, or in twos or threes; the colouration of the nuclei varies in intensity.

In the fibrous tissue at several spots in the immediate vicinity of an epithelial column are small collections of deeply coloured granules of varying sizes, mostly spheroidal in shape; in some of the alveoli are a few similar granules.

In a second specimen (seventh case previously cited) chromatic granules occur in the cell-protoplasm. From a nucleus here and there are to be observed a few distinctly stalked granules or

Woodcut 19.

Scirrhus mammæ put directly into Müller's fluid. *Not incubated.*

capitate processes which project into the cell-protoplasm, the border of the latter being quite distinct, polyhedral, and adapted to the adjoining epithelial cells of the column.

In a third specimen (eighth case before cited) coloured granules are to be seen in a few situations free in the alveoli and the fibrous tissue, some of them of a very delicate tailed form.

In a fourth specimen of mammary scirrhus from a widow, aged 57, the mother of three children, the disease was noticed in November, 1887, as following an injury in October; infiltrating kind; skin adherent; lymphatic glands diseased. Operation, February 4th, 1888. In the protoplasm of several of the epithelial cells occur comma-shaped granules of chromatin; and here and there epithelial cells amongst others of a group, the nuclei of which are quite devoid of coloured particles. Coloured granules are observable also in the fibrous tissue.

As an example of sarcoma experimented upon by the same method, we may adduce that of a typical spindle-celled sarcoma of the thyroid. The tumour was from a man aged 42. Repeated recurrence ensued after the first operation, and as many partial removals; the common carotid was eventually tied for hæmorrhage occurring from the growth, and the patient died April 7th, 1888. No secondary growths were found in the viscera.

In a logwood preparation made from a piece of the growth hardened in Müller's fluid and then in spirit the following appearances are seen: Nuclei of cells sharply defined, deeply

stained points of nucleoplasm in a more lightly-coloured nuclear matrix; they contain one or more nucleoli in places. Karyokinetic figures occur at rare intervals.

Leucocytes are present in fair abundance amongst the cells of the tumour; they are darkly and uniformly stained, of considerably smaller size than the nuclei of the tumour-cells, mostly spheroidal, but some are elongated. There is distinct budding from many of the latter elements, and from a few of the smaller nuclei of the tumour-cells.

There is in the section an area also of necrosis, in which no chromatic substance is seen except in the form of granules strewn in the tissue; the granules are of various shapes and sizes; some are of large size, and such are spherical; in these cases the fibrous tissue is also necrosed; in the cells bordering the disintegrating area the nuclei are unstained.

This disintegration of the nuclei is of no uncommon occurrence in portions of tumours that have undergone necrosis.

A piece after twenty-four hours' incubation on sheep's serum shows similar extrusion of the nucleoplasm of the nuclei, as described in carcinoma; here and there occur disconnected granules in the protoplasm, and free granules amongst the cells. There are no signs of degeneration in these parts.

A piece after forty-eight hours' incubation shows capitate chromatic processes projecting from a few of the nuclei into the cell-protoplasm, there being no signs of degeneration.

There is in this piece a necrotic area in which the nuclei are unstained, and the tissue thickly strewn with spherical granules of various sizes, some considerable, and derived doubtless from the nuclei of the necrosed cells.

A piece after seventy-two hours' incubation shows minute sessile buds of chromatin projecting from the margin of many of the smaller oval nuclei, there being in those parts none of the ordinary marks of degeneracy.

The appearances described we have not observed in normal tissues kept sterile on nutrient media, whether incubated at 100° F. or not. We may describe the following examples:

Muscle of a healthy dog (Plate XXXV, fig. 3), incubated eight days and subsequently hardened in Müller's fluid and then in spirit. Sections stained with fuchsin show remarkably little departure from the normal; the striation is clear in the fibres, in some the

transverse, in others the longitudinal being chiefly marked; it is not quite so sharp as in the normal. Nuclei of muscle-corpuscles oval for the most part, lightly stained plasma with darker points of matrix, not so sharply defined as the normal. Large granular plasma-cells occur in the connective tissue.

Muscle of healthy dog.—Incubated for eight days, kept in the tube altogether fifteen days; fuchsin stain. Striation indistinct, nuclei of muscle-corpuscles lightly stained with darker points so as to appear highly granular, contour regular, but less sharp than natural.

Muscle of healthy rabbit.—Kept in tube without incubation, for eight days. Transverse striation of fibres well marked; nuclei of muscle-corpuscles oval, sharply defined, granular.

Muscle of healthy rabbit.—Incubated eight days. Striation distinct in both directions; nuclei of muscle-corpuscles lightly stained with darkly coloured points, contour regular and distinct.

Muscle of healthy rabbit.—Kept without incubation for eight days and then incubated for eight days. Striation of fibres fairly well marked; nuclei of muscle-corpuscles very granular, contour of nucleus indistinguishable.

Kidney of healthy dog.—Incubated eight days. Nuclei of renal epithelium visible in some of the cells whilst in cells contiguous the nuclei are unstained; in others again the nucleus is recognisable as a very faintly stained granular spheroid. The disappearance of nucleus appears to result from the absence of coloration, the nuclear network becoming indistinguishable from the cell-protoplasm; the nuclear chromatin does not undergo dispersion; no free-coloured granules are observable in the cell-protoplasm or tissue around the tubules.

Kidney of healthy dog.—Incubated eight days, kept altogether fifteen days in the tube. Nuclei of epithelium in places well stained, in places barely visible, although in cells contiguous with the first. When stained, the nuclei are regular in form and granular; some of the nuclei are faintly stained, but wanting in sharpness of outline; in some cells no trace of nucleus is discoverable. Cell-protoplasm granular, faintly coloured.

Nuclei of connective-tissue cells of stroma well stained, granular as are also those of the capillary epithelium of the glomeruli.

Remarks.—Although we are at present unable to offer any evidence

of the real import of these remarkable appearances in cancer, we cannot help thinking that they may have an intimate bearing on the local and general infective processes of the disease.

We are acquainted with the disintegration into irregular masses of the nuclear substance which occurs in conditions of acute inflammatory necrosis, as seen in glanders, or such as is observed in the nuclei of pus-cells, or in portions of new growths in which degenerative softening has occurred, or when a tissue is subjected to unequal and excessive heating.

In the cutaneous lesions of glanders more particularly, we have observed the nuclei of the leucocytes remarkably irregular in form, divided into flask-shaped processes, mostly of relatively large size, which become detached and are found free in the surrounding tissue.

The process we have described in cancer has not, as compared with the above-mentioned cases, the appearance of being a passive cleavage or division of the nucleus ; in the epithelial cells especially the outline of the nucleus remains quite distinct and sharp, whilst the chromatic substance projects beyond.[1]

The possibility of these changes having an artificial production is of course not to be ignored, such as is known to obtain in the extrusion of the nucleus from the newt's blood-corpuscle under the influence of certain chemical agents, of the protagon from medullated nerve-fibres, and the like.

There are, however, among living processes some with which they bear comparison. One of these is the series of changes which takes place in the ovum independently of and preparatory to fertilisation.

In the maturation of the ovum there occurs an extrusion of the nuclear chromatin, with portion of the protoplasm from the cell beneath the vitelline membrane, the extruding part constituting one of the polar globules; by a repetition of this process a second globule may be extruded. The extrusion is not, according to the latest views, preceded by an ordinary karyo-kinetic process. The nucleus of the ovum, in which now no nucleoplasmic network is visible, remains as the female pronucleus, and the cell in this state is ready on fertilisation to undergo the segmentation involved in the production of an embryo. In explanation of the extension of the polar globules, Balfour has advanced the hypothesis that the

[1] Our impression is that the process occurs before the death of the tissue-elements, *i. e.* within a comparatively short period after the removal of the tumour.

ovum-cell is originally hermaphrodite, and that the process implies the extrusion of the male element, its end being the prevention of parthenogenesis by necessitating the entrance of germ plasma from a second individual, the male, in order that segmentation may proceed.

The subject of spermatogenesis has been worked out in the rat from a correlated point of view, by Mr. Herbert H. Brown, of University College, in a paper in the 'Quarterly Journal of Microscopical Science,' vol. xxv, 1885. The most interesting observations in this paper relate to the so-called seminal granules, which are usually stated to arise by disintegration of the nuclei of the innermost cells of the seminiferous tubuli. According to Brown, these granules arise from the developing spermatozoa, from which they separate off during the later stages of development, and he suggests that the process may represent the elimination of the female element from the spermatozoa, the spermatozoa, like the ova, being derived from cells originally hermaphrodite.

In the process of fertilisation the head of the spermatozoon penetrates the ovum-cell, and enlarges to constitute the male pronucleus; after the fusion of the male and female pronuclei the process of segmentation commences.

It is tempting, therefore, to imagine that the appearances in carcinoma may indicate the working of a sexual process, that the extrusion of the chromatin prepares the nucleus and cell for subdivision, whilst the extruded particles may represent the male elements of such cells, and incite division in others after the manner of spermatozoa. Should this prove to be so, the extruded particles might be regarded as cancer sperm and named carcinozoa, although the latter term would hardly, under the modern limitation, include similar elements arising in sarcoma.

The only observer to whom we need refer in connection with this subject is Gussenbauer ('Zeitschrift für Heilkunde,' Band ii, 1880).

Gussenbauer's long researches were limited to the secondary infection of lymphatic glands that occurs in carcinoma, and in some cases of sarcoma. And although he does not describe or figure the remarkable appearances observed by us in the primary tumour, his conclusions with regard to gland infection are in accord with the appearances described in the present communication.

Gussenbauer states that minute granules are to be observed in

infected lymphatic glands, free amongst the cells, and also within them, his interpretation of the appearances being that the granules in question are really the seminium or infecting particles which incite the cells to multiply and take on the characters of the primary growth.[1]

It must be confessed, in criticism, that these appearances figured by Gussenbauer admit of another interpretation. It is possible that the appearance of a growth of epithelium out of elements which look like lymph-cells may be due to rapid multiplication of transported epithelium, the new and young elements being at this stage not sufficiently differentiated to admit of their being distinguished from the indifferent cells of the surrounding lymphatic tissue. And the granules mentioned by Gussenbauer, though carried by lymphatic channels from the alveoli of the carcinoma to the glands, may, in place of actively entering the cells of the latter and inciting them to an atypical division, be granules taken within the cells as are inert particles under similar circumstances. It is right to state, however, that Gussenbauer thinks that the granules, though infective, are in some cases destroyed by the lymph-cells, though the evidence which he adduces of this is not strong, viz. that clinically a temporary enlargement of glands is witnessed, independently of an inflammatory process in the primary tumour, the glandular affection not proceeding to metastatic formation.

If the granules in question have anything to do with the production of the secondary tumours it is easier to imagine this to be by inciting division in cells transported from the primary tumour to the gland than by inciting lymph-cells and the cells of the blood-vessels (Gussenbauer, loc. cit.) to undergo transformation into squamous-celled, columnar, or other epithelium.

May 15*th*, 1888.

Addendum.—The illustrations on Plate XXXIV are introduced to record the negative results obtained by incubating portions of new formations, which we recorded in the preceding volume of the Society's 'Transactions.' Fig. 1 shows a portion of a spindle-

[1] Mr. Butlin informs us that a number of sections of cancerous glands were prepared for him with great care by Mr. A. G. Francis and Mr. Lyndon, of St. Bartholomew's Hospital. The method described in the 'Zeitschrift für Heilkunde' (ii, 17) was closely followed, but he failed entirely to discover the characteristic molecules figured by Gussenbauer.

DESCRIPTION OF PLATE XXXIV.

Illustrating Messrs. Shattock's and Ballance's note on the Histology of Cancer, &c., after Incubation. (Page 409.)

From drawings by Mr. Shattock.

FIG. 1.—Portion of a sarcoma sterile after incubation at 100° F. in nutrient gelatine.

FIG. 2.—Portion of scirrhus of breast sterile after incubation at 100° F. on agar.

DESCRIPTION OF PLATE XXXV.

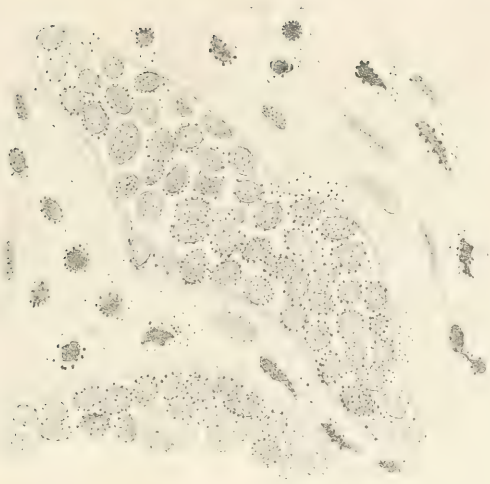
Illustrating Messrs. Shattock's and Ballance's note on the Histology of Cancer, &c., after Incubation. (Page 409.)

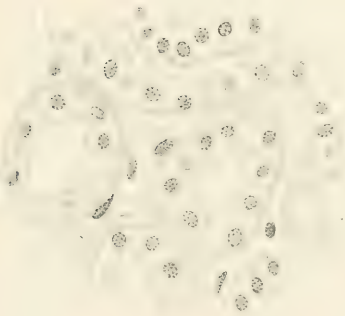
From drawings by Mr. Shattock.

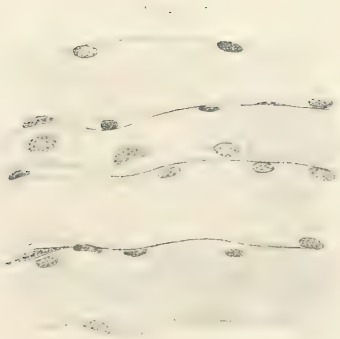
FIG. 1.—Section of scirrhous carcinoma of breast made from a portion incubated on solidified blood serum (sheep) for eight days at 100 F., showing the extrusion of the nuclear chromatin in the form of granules from the nuclei of the epithelial cells and connective-tissue corpuscles.

FIG. 2.—Section of healthy dog's kidney made from a portion incubated for eight days at 100° F. on solidified blood serum.

FIG. 3.—Section of healthy dog's muscle made from a portion incubated for eight days at 100° F. on beef peptone agar.







celled sarcoma of the thyroid, removed July 6th, 1887, incubated in nutrient gelatine for eight days at 100° F., and kept subsequently for some time at an ordinary room temperature. Fig. 2 shows a large piece of a mammary scirrhus, removed May 12th, 1887, incubated on agar for five days at 100° F., and then left at the ordinary temperature. At the present date (October, 1888) it has undergone not the least perceptible change; the end of the tube is hermetically sealed with gutta percha. In neither case did any growth of micro-organisms occur.

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3. *On hæmorrhagic infarction of the liver.*

By L. C. Wooldridge, M.D.

Hitherto it has not been possible to obtain experimentally a hæmorrhagic infarction of the liver. The branches of the portal vein are strictly analogous to an end artery, but owing to the fact that the hepatic artery breaks up into capillaries which anastomose with those of the portal vein, the occlusion of the branches of the latter by emboli is not followed by any hæmorrhagic infarct.

Thus Cohnheim and Litten found that large quantities of finely divided inert bodies could be injected into a mesenteric vein without producing any hæmorrhagic infarction of the liver, although the obstruction in the portal vein was such that ascites was set up.

In human pathology a simple hæmorrhagic infarction is, I believe, a rare or almost unknown occurrence. In the present note I shall describe an experimental method of inducing the most widespread infarction of the liver, and I hope to show that some of the commonest pathological changes in the liver possibly originate by this or an allied process.

The experimental process to which I allude consists in injecting a small quantity of a solution of a substance into the jugular vein of the dog. This substance is a complex proteid body which can be readily obtained from various tissues, particularly the thymus gland. It can, however, be got from many other sources, as liver,

brain, testis, &c. I have described[1] the preparation and properties of this substance elsewhere, and need not repeat them here.

If a solution of this substance be injected into the jugular vein of a rabbit the animal dies instantly, owing to complete intravascular clotting of the blood. If, however, it be injected into the jugular vein of the dog it only causes clotting in the portal vein, its tributaries and its branches. That is to say, if some of the substance be injected into a dog's jugular a clot is invariably formed in the portal vein. The amount of clot formed varies with the amount of substance injected. If the latter be large the whole portal system, from the finest divisions of the mesenteric veins and to the terminal branches of the portal vein in the liver, is completely thrombosed. In these cases, where a large amount is injected, the animal generally dies at once, and frequently a small amount of clot is found in the right heart.

But by injecting smaller quantities it is readily possible to induce a very extensive thrombosis of the portal vein without killing the animal and without producing any appreciable clot anywhere else. If the animal be killed a few days after the injection a clot is always found in the portal vein, and the liver shows more or less extensive infarctions.

This is the method. I will now describe, first, the early changes in the liver, and subsequently the later. The early changes are hæmorrhagic in character, and since they occur as the result of a process of thrombosis they may be classed as hæmorrhagic infarction, although, as I shall discuss later, it is questionable whether the process is identical with the ordinary infarction that one gets in the liver or spleen. The hæmorrhages partly occur in well-defined more or less pyramidal areas which have a deep red colour, and contrast strongly with the pale colour of the rest of the liver.

A microscopical section shows the capillaries of this area to be enormously distended with blood, whilst large actual hæmorrhages, both in the substance of the lobules and in the portal canals, are seen. But in addition to these patches, or in place of them, a diffuse hæmorrhage all over the surface of the liver may be met with, and the blood may be collected into large blebs beneath the capsule of the liver. A very peculiar fact must here be noticed, with regard to the gall-bladder. It is especially common, after

[1] 'Proceed. Royal Society,' 1886.

injection of even very small quantities of the proteid, to find most extensive extravasation of blood beneath the peritoneal covering of the gall-bladder. I noticed in one case in which this was very marked that the gall-bladder itself was filled with a colourless mucus of the most extreme tenacity; the mucus was just passing into the common duct, which it must inevitably have obstructed. It is possible that in this way slight changes in the blood may give rise to jaundice. I have noticed this hæmorrhagic affection of the gall-bladder as the result of the injection into one dog of blood-plasma from another dog. It may be noted that ascites is never produced as the result of the plugging of the portal vein, no matter how complete the latter may be.

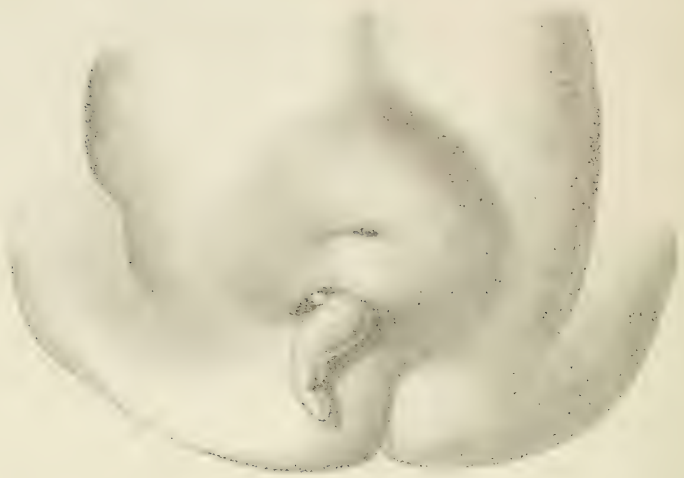
The later changes in the liver as the result of this injection are as follows: In the first place the clot in the portal vein has always completely disappeared, after an interval of fourteen days and probably much less. After this interval there is not the slightest trace of a clot to be found either in the main trunk of the portal or its branches. The liver may be apparently but little altered, except for some slight thickening of the capsule here and there in patches. Careful microscopical examination of these livers always reveals, however, scattered patches of early cirrhosis.

In other cases the whole liver shows the most profound structural changes,—in an earlier stage large patches of what I regard as coagulation necrosis of the liver tissue; in a later, the most extreme fatty metamorphosis, together with less marked cirrhosis.

The very striking difference between my results and those of Cohnheim and Litten is only, I think, capable of the following explanation: I may first mention as a preliminary that no thrombus is discoverable in the hepatic artery as the result of the injection. We speak at present of two kinds of emboli or thrombi, simple and infecting. The thrombus produced in the portal vein by the method described is neither a simple inert thrombus, for the latter produces no infarction, nor is it infective, for it produces no abscesses. Its specific character lies in the fact that it gives rise to most extensive hæmorrhages. The injection of the proteid substance used in these experiments not only causes clotting but also induces very marked chemical changes in the blood. In fact, there is set up a temporary and mild sort of hæmorrhagic diathesis. The blood which is drawn off after the injection does not clot, or only very slowly and imperfectly. Where there is a

local injury extravasations of blood are liable to occur. Thus there is always considerable hæmorrhagic infiltration of the tissues in the neighbourhood of the wound, which latter is of a most trivial character. I think it is the coincidence of these two factors, the hæmorrhagic condition of the blood and the thrombosis, which produces the infarction of the liver. As described above, the later changes in the liver are principally those which we are accustomed to speak of as chronic inflammatory changes, and perhaps these observations may throw some light on the origin of these very obscure processes.

This paper is only intended as a preliminary communication. A complete discussion of the observations recorded here would of course require much more of the time of the Society than I should feel justified in taking up. Specimens of the clot in the portal vein, of the infarction of the liver, and of the cirrhosis and fatty changes are shown. *November* 1*st*, 1887.



Ada Stone.

DESCRIPTION OF PLATE XXXVI.

Fig. 1 illustrates Mr. E. Owen's case of Sacral Tumour with Anomalous Appendage. (Page 425.)

Fig. 2 illustrates Mr. Stonham's specimen of Cyst of the Peritoneum. (Page 149.)

XI. MISCELLANEOUS SPECIMENS.

1. *A case of sacral tumour with anomalous appendage.*

By Edmund Owen (introduced by F. Penrose, M.D.).

[With Plate XXXVI, fig. 1.]

B. H—, aged 11 weeks, under the care of Mr. Edmund Owen at the Hospital for Sick Children, Great Ormond Street, to whom it was sent by Dr. Fletcher, of Charterhouse Square.

The subject is a well-nourished baby with the following deformities:

On the lower part of the back is a rounded swelling of the following dimensions:

From above down	$3\frac{3}{4}$ inches
From side to side	$3\frac{1}{4}$ inches
From before back about . . .	$\frac{7}{8}$ inches.

The lower limit is just above the vertical crease of the nates. It is very nearly, but not absolutely, in the middle line, being a little to the left side.

In the exact centre of the surface of the swelling is an umbilication a quarter of an inch in depth.

Half-way between this dimple and the lower border of the swelling, but slightly to the left of the middle line, is an appendage (inclining to the left) two inches in length.

Although the general inclination of the appendage is to the left, the tip curves slightly round towards the right, owing to two constrictions, most marked on the right side, occurring in the length of the appendage, and giving it the appearance of a fat "little finger."

The base of the appendage is constricted; the greatest circumference is one third of an inch from the point of attachment to the general swelling.

On the left side of the base of the appendage is a second small excrescence about a quarter of an inch in length.

A line drawn from the umbilication to the fold between the nates would not pass through the appendage, but would fall just to the right of it.

The swelling and appendage apparently consist entirely of soft elements, and the consistence and appearance are, perhaps, more like Wharton's tissue, as found in the umbilical cord, than like fat. There is no fluctuation.

No malformation of the spinal column can be definitely made out, but there is a very ill-defined vertical ridge deep down beneath the central umbilication, which, together with the umbilication, is suggestive of spina bifida.

There is flexion of both hands at a right angle with the forearm together with flexion of the digits, so that the finger-nails occasionally touch the forearm.

In the left hand the middle and ring fingers are most pressed in towards the forearm, the index and little fingers meeting behind them. The thumb is pressed on to the palm, which is concave and narrowed from side to side; the thenar and hypothenar eminences approach each other.

In the right hand the arrangement of the fingers is slightly different.

The middle finger is most pressed in towards the forearm, so that from the dorsal surface only the proximal half of the first phalanx of the digit can be seen, the rest of the finger being completely hidden by the meeting of the index and ring fingers behind it. The little finger is less flexed than the others. The thumb is completely pressed into the palm.

Both hands are badly developed, being much smaller than natural.

There is no other abnormality detected; no morbus cordis; no evidence of congenital syphilis.

Past history.—Full-term child. Mother in labour three days; no instruments were used. The mother gives no history of fright or fall during her pregnancy; she was in good health and quite steady all the time, and was never short of food.

Family history.—Father, a labourer, aged 27; no history of phthisis, rheumatism, or syphilis. Mother aged 26; no history of phthisis or rheumatism; she has had three children, of whom the

eldest was ill from its birth, and died when four months old of "inflammation of the chest;" second is living and quite healthy; third is the patient. The mother has had no miscarriages.

As regards the nature of the rounded swelling and the appendage, I will suggest that the former is not unlikely the result of spina bifida, whilst the appendage may be, and possibly the rounded tumour as well, the result of a very imperfect attempt to produce a double monster. The sacral region is by no means an infrequent site for the attachment of a fairly developed or a rudimentary fœtus. *February 7th*, 1888.

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2. *Report on a living specimen of parasitic fœtus.*

By J. Bland Sutton and Samuel G. Shattock.

Drawn up under the sanction of the Society.

[With Plate XXXVII.]

Certain details of the following report are taken from a description given about two years ago by Dr. R. M. Khory, M.D.

The boy (Laloo) shown this evening at the Society, and who is now being publicly exhibited in London, is aged 17, and a native of Oovon, in Oudh. The child was born in the cranial position, the hands of the parasite encircling his neck. His mother has borne four children; the first, now aged 20, is a female. Both father and mother are strong and healthy; their respective ages are 50 and 44. The patient is the second child; two other sons were born after him, one three years after the birth of Laloo, the other five years after the birth of the third.

The third and fourth children are now dead; the third died at the age of three years, and the fourth four months after its birth. There is no family history of malformation.

Present condition.—Laloo is 5 ft. 1½ in. in height, and a well-developed, intelligent, good-looking boy. His general health is excellent. When about two years old he suffered from smallpox the eruption appeared on the parasite as well as on the patient

and typical scars are still visible on the left leg and both the upper extremities of the former.

Description of the parasite.—The parasite, as it appears externally, consists of two main divisions, a superior, constituted by the upper extremities, and an inferior, larger, division which includes the pelvis with the genital organs and lower limbs. Each of these is attached to the autosite by a constricted portion or pedicle. As the posterior surface of the parasite looks forwards its right side will, in description, correspond with the left side of the boy; the usual anatomical rule in this respect is adopted throughout the report.

A. *Description of the upper division of the parasite.*

The pedicle of this arises in the middle line, three inches below the level of the nipple, and is composed of a firm, central process of bone invested with lax integument; the pedicle is conical, at its base it is 3 cm. in diameter, and its length is 5 cm. (2 inches about). By its base the bone is combined with the lower part of the thoracic wall to the right of the middle line; and moveably articulated to the summit of the pedicle is a wedge-shaped osseous mass representing the combined scapulæ. This has a circumference of 1·7 decimeter ($7\frac{3}{4}$ inches about); from its anterior aspect there projects a single median cylindrical process of bone.

There are two nipples on the skin over the scapulæ, 6 cm. apart, and 3 cm. from the base of the pedicle.

The humeri are well developed, and a fair amount of movement is allowable at the shoulder-joints; the upper arms can be brought together and mutually withdrawn to an extent that permits of the hands being placed on the boy's shoulders. Both the elbow-joints are immovably ankylosed.

Left upper extremity.—The carpus is small but distinctly palpable; it is somewhat flexed and admits of slight passive movement. The fingers are slightly webbed; the thumb is drawn towards the palm.

The limb measures from the shoulder to the tip of the middle finger twenty-one inches (the recorded measurement two years ago was seventeen inches).

The forearm, from the extreme point of the elbow to the wrist, is six inches; its circumference at a mid-point is three inches.

Right upper extremity.—The forearm is considerably smaller on

DESCRIPTION OF PLATE XXXVII

To illustrate the report by Messrs. J, Bland Sutton and S. G. Shattock on a Living Specimen of Parasitic Fœtus. (Page 427.)

From photographs.

this side than on the left; there is but a single cylindrical bone to be felt in it. Carpus well developed; hand abducted; thumb absent. The shortness of the forearm has apparently resulted from an injury which was received early in life.

The limb measures from the shoulder to the tip of the middle finger fourteen and a quarter inches (the measurement recorded two years ago was twelve inches). The forearm is two and three-quarter inches in length and three and a quarter inches in circumference.

The upper limbs of the boy measure, from the shoulder to the tip of the middle finger, twenty-nine inches (two years ago the measurement was twenty-four, inches), the limb being placed in a position the same as that of the left longer upper extremity of the parasite, viz. with the elbow at an obtuse angle.

When the boy stands, the upper limbs of the parasite lie pendent to the left side, against the left side of the abdomen of the boy and upper part of the left thigh; together with the conjoined scapulæ they admit of a complete rotation in a vertical transverse plane.

B. *Description of the lower division of the parasite.*

The pelvis is, like the conjoined scapulæ, attached to the autosite by a well-marked pedicle. The pedicle contains, however, no bone; its circumference is 13 cm., and its length 4 cm., and it is attached at a point two inches below and somewhat to the right side (boy's) of the upper. There is to be felt in it a firm, pliable, apparently fibrous band about as thick as the finger. On compresssing the pedicle between the thumb and fingers, intestinal gurgling is very obvious, but no pulsation can be detected in it.

The lower division is constituted by an ill-formed pelvis and two lower limbs. A sacrum may be made out in the middle line, and above and behind this the hip-bones appear to be approximated; the latter are traceable in front to a pubic symphysis.

There are two symmetrical gluteal folds. The anus is imperforate, its situation is marked by a shining linear scar. There is a well-formed penis, and the pubic hair is abundant; no testicles are to be felt.

Both the hip-joints are ankylosed in a position of semi-flexion. The knees are flexed at a slightly acute angle. No movement is allowable; and very little at the ankle-joints, which are fixed at a

right angle. Behind the knees the integument forms a broad, dense, falciform fold. An external and an internal malleolus are present in both limbs, but no distinct fibula can be felt.

The length of each of the lower limbs of the parasite, from the hip to the dorsum of the foot, and thence to the tip of the middle toe, is twenty-seven and three quarter inches (two years ago it was twenty-four inches).

The length of the lower limbs of the boy, taken in the same way, is thirty-eight and a half inches (two years ago, thirty-six inches).

The left foot has but three perfect digits, the terminal phalanges being absent in the first and second. There are scars of old burns on the soles of both feet, and on the left buttock.

Physiology of the parasite.

Movement.—No spontaneous movement occurs, nor can the boy will to move any portion of the parasite, which has at no time been capable of any but passive movement.

Sensation.—Light touches with the point of a quill pen are not felt; more sharply inflicted ones are felt at all parts, and can be localised by the boy when blindfolded. The upper limbs are more sensitive than the lower.

The parasite perspires simultaneously with the boy, and its skin becomes hot when the boy feels feverish.

Urine is passed independently by the parasite, and without the boy's cognisance. After a heavy meal the boy complains of a feeling of weight in the parasite. Erection of the penis of the parasite at times occurs.

Dr. Coupland was unable to detect any heart-sounds or pulse in any of the limbs of the parasite.

The surface temperature, taken between the gluteal folds, is below 95° F., *i. e.* lower than the clinical thermometer registered.

A few days after the drawing up of the report, Mr. Sutton was asked to see him, and found that a large piece of intestine had descended into the parasite and was strangulated. This was reduced, and a truss designed to prevent a recurrence of the accident.

The case under notice corresponds very closely with the well-known Chinese, Ake, of whom a model and a description are in the Museum of the Royal College of Surgeons.

In Geoffroy St. Hilaire's work ('Anomalies de l'Organisation') it is further stated, in regard to the physiology of the parasite in the latter example, that there was no spontaneous movement, and that when the parasite was pinched or pricked a little strongly the autosite experienced pain and was able to localise it. The temperature of the parasite was normal. That author refers to three other examples of similar malformation. Two were infants, and the third was a man who became the father of a daughter and three sons, all of whom were well formed and of robust health. One of the infants was a female.

In all these cases micturition occurred independently in the parasite, the rectum was imperforate, and in most, strong pinching of the parasite could be felt by the autosite.

Foerster ('Missbildungen des Menschen') makes three divisions of parasitic fœtus connected with the thorax and epigastrium. In the first, the parasite has head, trunk, and limbs; in the second it is acephalous; in the third a trunkless head alone exists.

Of the first kind the most perfect example is that of Lazarus Colloredo, who was aged 28 and born in 1716; the parasite was wanting in the lower extremity. Foerster states that of this kind he has collected seven instances.

Of the second, acephalous, class, to which the specimen under notice belongs, he collects thirty-five cases, twenty-three male, four female. These cases include those already quoted from Geoffroy St. Hilaire. That recorded by Ambrose Paré also comes under this head.

"In the year 1530 there was a man to be seen at Paris out of whose body another, perfect in all his members except head, hangeth forth as if he had been grafted there. The man was fifty years old, and he carried the other, implanted or growing out of him, in his arms, with such admiration to the beholders that many ran very earnestly to see him" (Works of A. Paré). Paré's description of the case is illustrated by a drawing which is reproduced by Witkowski in his 'Histoire des Accouchements.'

Foerster remarks generally of this group that the anus is always closed, and that the genital organs are mostly wanting or have blind canals when present; the kidneys may be present, rudimentary, or absent; the bladder is seldom wanting; the urethra usually impervious but at times open and allowing urine to dribble away; the internal generative organs mostly rudimentary.

Foerster's third division includes trunkless parasites, in which there is a head and rudimentary neck and thorax alone present; he notes five examples.

In regard to the production of the malformation under notice there are two views. One view is, that an originally single ovum gives origin during its development to two embryos, *i. e.* that the germinal area undergoes cleavage, the yolk-sac remaining common to the two parts, the rate of growth in one part subsequently falling short of that in the other.

The second view is that two distinct ova are concerned, one of which becomes at an early stage incorporated with, or included, in the other.

In the present instance the latter view would not explain the formation of an alimentary canal common to the parasite and autosite, which is readily explicable, however, on the first.

The fact that in all the recorded cases the autosite and parasite are of the same sex is also an argument for the first view rather than for the second; to this may be added the autosite's perception of sensation in the parasite, which fact points to the singleness in origin of the sensorium.

February 21*st*, 1888.

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3. *A fœtus with spina bifida occulta and some remarkable associated abnormalities.*

By J. Bland Sutton.

A few months ago a fœtus, born between the eighth and ninth months of gestation, came into my possession, which presented a large number of abnormalities. The defects in themselves were but little out of the common, and a collection of them in one fœtus is not unusual, but in the present case, it is the association of certain lesions that establishes the value of the specimen from a pathological point of view.

Externally the most marked features were absence of the anus, lateral curvature of the spine, the convexity being to the right, the curve involving the whole spine; the left foot was in the position

of talipes equino-varus, and the leg was slightly rotated on the femur. The position of the foot was suggestive of some central lesion, and on removing the skin of the lumbar region some of the vertebral arches were found to be defective, although nothing abnormal could be detected by manipulation through the skin. The condition of the spine will be dealt with later.

On eviscerating the fœtus, the left kidney was absent; no renal artery or ureter could be distinguished on that side. The corresponding suprarenal body and testicle were quite normal in size and position. The right kidney was of the usual size, a state of things somewhat rare in instances of single kidney.

The rectum, enormously dilated and filled with meconium, ended as a cul-de-sac at the prostate, but a narrow duct could be traced through its substance to terminate in a mass of muscle-fibre in the raphe of the perinæum, the situation of the normal anus.

The cæcum was short and blunted, the vermiform appendix being represented by a fibrous band.

The pharynx was imperforate, and the upper end of the œsophagus communicated by an oval orifice with the trachea. The upper end of the œsophagus and the lower end of the pharynx, however, were united by a short but wide band of fibrous tissue. The mouth, pharynx, larynx and tongue were in all other respects normal.

From a perusal of the accounts of similar malformations there is good reason to believe that, in the majority of imperforate pharynges, the upper extremity of the œsophagus communicates with the trachea as in this specimen. Summarising the defects in the alimentary tract we have: 1, an imperforate pharynx; 2, a communication between the œsophagus and trachea immediately below the fifth ring; 3, fibrous degeneration of the vermiform appendix and abnormal shortness of the cæcum; and, 4, imperforate anus.

We must now deal with the vertebral column. On dissecting the soft parts from the lumbar region it was evident that we had to deal with a *spina bifida occulta*, that is to say, the laminæ and spines of some of the vertebræ were defective, but no cyst protruded externally, and the skin covering the spot was natural. The imperfect arches corresponded to the fifth lumbar, the second and third sacral vertebræ, the defective spots being covered in by very dense fibrous tissue.

The arches were bisected, and the cord examined, and it turned

out that the cord, which, in the fœtus at birth normally terminates opposite the centrum of the second lumbar vertebra, in this case extended to the tip of the coccyx, where it ended in a fatty swelling of the size of a large nut. Opposite the defective arches the central canal of the cord presented a dilatation of about an inch in length and capable of admitting easily the blunt point of a cedar pencil.

After the cord had been removed a more critical examination of the spine became possible, and some points of considerable value came to light. Between the eleventh and twelfth dorsal vertebræ a supernumerary half-vertebra existed. Between the twelfth dorsal

Woodcut 20.

Supernumerary half-vertebræ.

and first lumbar another supernumerary half-vertebra, much larger than the first one, was found, and as this occurred on the right side it readily explains the curvature of the column having its convexity to that side. The fifth lumbar and the first two sacral vertebræ presented a variation of very great interest, as they shed important light on the subject of spondylolisthesis.

It is well known, as Dr. Neugebauer, jun., has pointed out, that in spondylolisthesis the fifth lumbar vertebra may exhibit a solution of continuity in the arch. The rupture in the arch occurs immediately behind the superior articular processes. Some writers are of opinion that this dissociation is secondary to the spondylolisthesis, whilst Neugebauer inclines to the opinion that there may be a congenital defect at this spot. The other alternative is, that

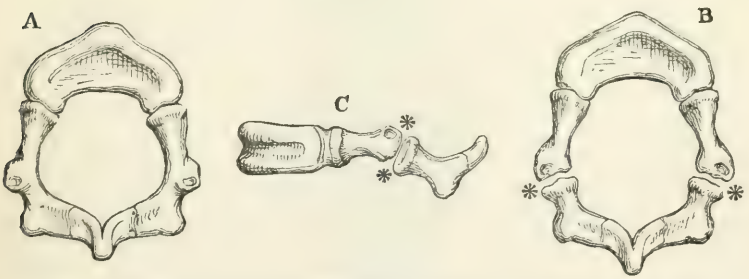
the absence of osseous continuity may be due to traumatism, followed by imperfect reparation, and the spondylolisthesis is secondary to the defect in the arch. Of course the great drawback to the argument that the defect is congenital exists in the fact, that, hitherto, the existence of a suture at this spot is at variance with our knowledge of the development of the vertebral arches. The only lines of union known are the neuro-central sutures which exist at the spot where the pedicles join the centrum (Woodcut 21, A.)

An attentive dissection of this spine has revealed to me the fact that the fifth lumbar and upper two sacral vertebræ exhibit false joints in the lamina. Thus on referring to Woodcut 21, B and C, it will be seen that behind the future articular processes there is a solution in the continuity of the arch. In two places perfect articulations exist, whilst in others the individual parts are connected by fibrous tissue. Hence this variation in the mode of development of these arches in this specimen comes as strong evidence in support of Neugebauer's view.

It may be mentioned that a congenital gap at this spot does not violate any morphological law, for in certain low vertebrate forms, the arch normally develops in this way.

Apart from the interesting variation in the ossification of some of the vertebræ it is also important to remember that the spina

Woodcut 21.



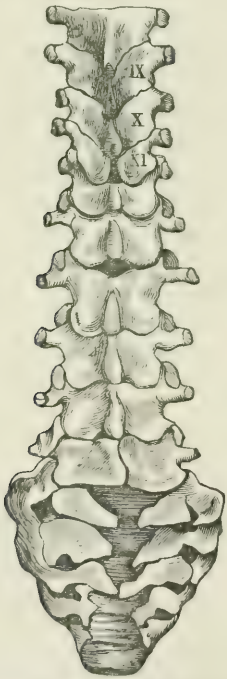
Three diagrams representing variations in the mode of ossification of the arches of vertebræ. A. Normal. B. The arch made up of two lateral pieces. C. Side view of the same.

bifida in this case most closely resembles the variety known as syringo-myelocele. In 1886 von Recklinghausen published an

elaborate research, entitled, 'Untersuchungen über die Spina Bifida,'[1] containing, among other things, descriptions of a number of cases of spina bifida, in fœtuses, associated with various defects in the alimentary canal, such as umbilical hernia, atresia ani, &c. Several of these were examples of spina bifida occulta. This association of defects of the spinal cord, especially syringo-myelocele, with abnormalities of the intestines is something more than casual.

A careful perusal of Recklinghausen's memoir, and a study of the fœtus which forms the subject of this paper, have furnished the last links in a long chain of evidence which convinces me that the spinal cord must be regarded as a highly differentiated section of

Woodcut 22.



The lower portion of a spine from a child affected with spina bifida occulta.

the primitive alimentary tract. Its central canal was originally part and parcel of this system, but is now functionally useless, and,

[1] Virchow's Archiv,' Bd. 106, 1886.

like obsolete canals in general, is frequently the seat of cystic dilatation-syringo-myelocele.

As an appendix to this case I will describe a spinal column which came under my observation several years ago. It is the spine of a child two years and a half old which I had prepared for teaching purposes. During its preparation my attention was arrested by a deficiency in some of the arches. The parts were carefully preserved for future study. A re-examination of the specimen shows that the spinous process of the tenth dorsal vertebra is rudimentary, that of the eleventh is wanting and the laminæ have failed to unite, thus leaving a gap in the spine. The spinous process of the twelfth dorsal and first lumbar are rudimentary. There is a similar but larger defect in the upper sacral region (see Woodcut 22). The case was probably one of spina bifida occulta. *November 1st*, 1887.

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4. *Mammæ in ovarian dermoids.*

By J. Bland Sutton.

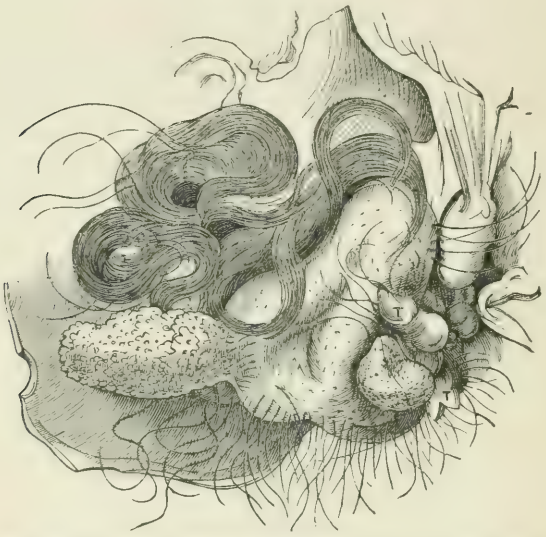
It is by no means uncommon to find in the interior of an ovarian dermoid one or more tags of skin resembling a nipple. Such nipple-shaped processes of skin may be associated with more or less rounded projections recalling in a striking manner the shrunken mammæ of a woman who has suckled many children. The surfaces of such mammæ occurring in dermoid cysts are dotted over with numerous small pits, indicating the orifices of sebaceous glands, and a crop of hairs usually sprouts from the skin-covered surface.

Such a specimen is represented in Woodcut 23, associated with teeth and locks of curly light brown hair. The surface of these nipple-like processes of skin is dotted over with numerous small pits, indicating the orifices of sebaceous glands. The specimen was removed by Sir Spencer Wells in 1863 from a young mulatto lady, and is preserved in the Museum of the Royal College of Surgeons.

Standing on the same shelf, besides this specimen, is a dermoid containing a mamma of the size of a walnut furnished with a

nipple. It is covered with dark-coloured, fine hair, and dotted with minute depressions which are the openings of sebaceous

Woodcut 23.



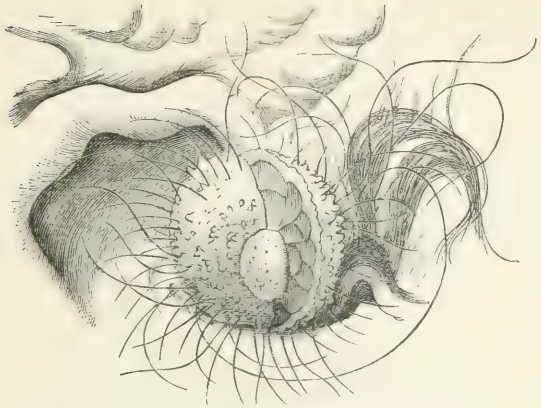
A shrunken mamma with an elongated nipple, from an ovarian dermoid. The cyst contained also hair and teeth. (Mus. R. Coll. of Surgeons).

glands. In minute structure it is made up of fat, covered with skin, having sebaceous and sudoriparous glands and all other characters of the integument (Woodcut 24).

The museum of the Middlesex Hospital contains an ovarian dermoid which has long excited my attention. It is a tumour of the size of an orange, and standing from its wall is a mass of fat covered with piliferous skin and furnished with a nipple. The mamma itself is supported upon a piece of skin-covered bone furnished with four teeth.

On section, the interior of the mamma was found to be occupied by a series of ducts leading to a glandular mass at its base. Unfortunately, the specimen had been many years in a preservative medium of the composition of which I am ignorant, but it has not preserved the tissues sufficiently for histological purposes, and this prevented me ascertaining the characters of the

Woodcut 24.



A mamma from an ovarian dermoid. A piece has been taken out for histological purposes. (Mus. R. Coll. of Surgeons.)

epithelium lining the acini and ducts. The mamma is shown entire and in section in Woodcut 25.

Dr. Granville Bantock has recently placed at my disposal some interesting ovarian dermoids; among them was one which presented the following characters: The cyst was the size of a melon, and was filled with the usual pultaceous matter containing shed hairs. Standing from the wall of this cyst was a rounded mass, of the size of a Tangerine orange, covered with skin furnished with a few short hairs. Passing from each side of this protuberance were two cylindrical processes covered with skin, attached by their distal ends to the cyst wall. On examining the parts more closely, the general resemblance of the mass to a mamma was very striking, more especially as the base of the larger of the two cylindrical processes was surrounded by an areola. On dividing these processes they were found to be hollow and full of fluid resembling very poor but viscid milk. Under the microscope this fluid had all the characters of milk and contained some cells resembling colostrum.

On dissecting the mamma it was found to be made up of a mass of fat of a rich yellow colour, traversed in its middle by a series of ducts which ended in a mass of glandular tissue lying at its base. It now became clear that we had to deal with two mammæ, with a common glandular mass. On examining the gland-tissue under the

microscope it was found to be made up of ducts, duct-like passages, and acini, arranged as in a normal mammary gland, from which they only differed in the character of the epithelium. In the

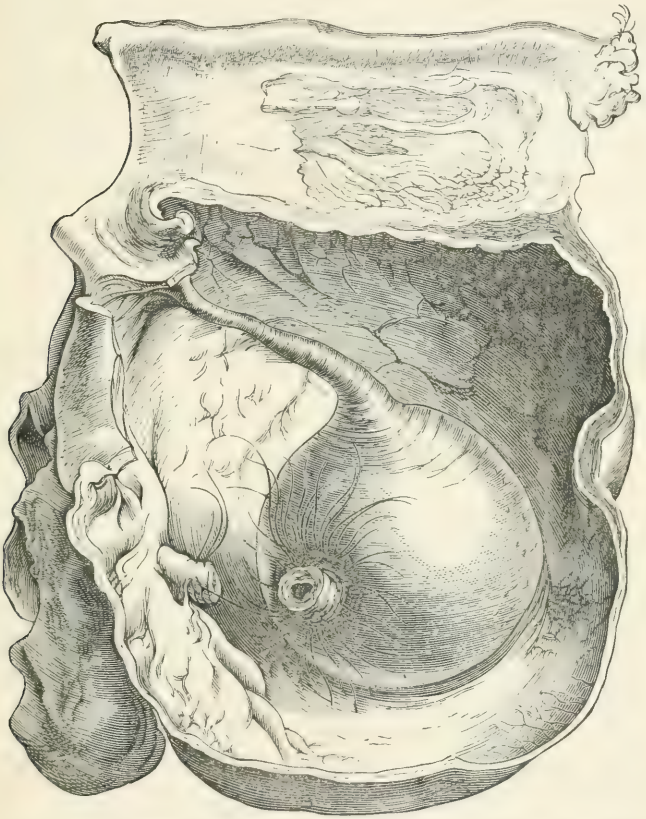
Woodcut 25.

A mamma from an ovarian dermoid. The upper figure shows its configuration and relation to the piece of bone with three teeth. The lower shows it in section, so as to display the disposition of the glandular tissue.

normal mamma the ducts and glandular recesses are lined with cubical epithelium; in this dermoid mamma the cells were several rows deep and spheroidal in shape.

The nipple differed from that of a normal mamma in possessing only a single duct, and this was large enough to admit a crow-quill. This may be explained by the fact that no outlet existed for the secretion, which had in consequence dilated the duct. Situated in the midst of the secreting tissue of this mamma were

Woodcut 26.



An ovarian dermoid containing a mammary gland furnished with two elongated nipples.

some nodules of cartilage with remarkably large cells, many of which presented calcareous deposits in their interior.

In these specimens we are able to trace every step, from a nipple-

like tag of skin to a mammary gland capable of furnishing a secretion resembling milk. The condition too is of some value inasmuch as it affords additional evidence in support of the opinion that mammary glands are modifications of sebaceous glands. These mammæ are interesting in another respect; not only do they resemble normal mammæ in shape, appearance, and, in their most complete form, in structure, but they seem to exhibit a tendency to form cysts in connection with the glandular part of their organisation resembling adenoceles or galactoceles.

'Virchow's Archiv,' Bd. 107, S. 505, contains a description of a mammiferous dermoid by Dr. Desiderius von Velits, of Budapest. In this case a well-developed mamma was present, and the ducts had a definite lining of cubical epithelium. *May* 1*st*, 1888.

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5. *Mammiferous dermoid cysts of the ovary.* (*Card specimen.*)

By Samuel G. Shattock.

[With Plate XXXVIII, fig. 2.]

The two specimens were successfully removed by Dr. Cullingworth (to whom I am indebted for leave to exhibit them), from a woman aged 39. The patient had borne twelve living children and had had three miscarriages. The youngest child is eighteen months, and the last miscarriage occurred three months ago. Swelling was only noticed within the last three months, *i. e.* since the last miscarriage.

There was a very extensive series of intimate adhesions between the small intestine and the tumours, which were removed with the two ovaries in which they grew.

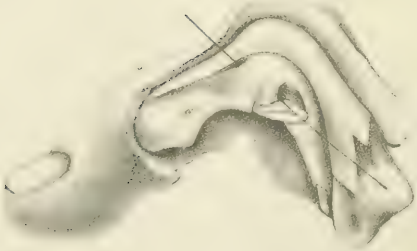
Description of the larger polycystic tumour.

The upper of the cysts, which is that bearing a nipple, is 3·5 cm. in diameter, and is in relation with the fimbriated end of the tube. Below this is an elongated, bilobed, more thickly walled cyst, 7 cm. in its longer, horizontal diameter. This contains, growing from its

Fig. 1

Fig. 2.

Fig. 3.



DESCRIPTION OF PLATE XXXVIII.

From drawings by Mr. Shattock.

Fig. 1 illustrates Mr. Shattock's specimen of Cyst in the Corpus Luteum. (Page 218.)

Fig. 2 illustrates Mr. Shattock's specimen of Mammiferous Dermoid Cysts of the Ovary. (Page 442.)

Fig. 3 illustrates Mr. Shattock's specimen of Hammer Toe. (Page 449.)

wall, a well-formed incisor tooth. Below this, again, is a much larger bilocular cyst, measuring 1·4 decimetres in its longer axis, and 9 cm. in its shorter.

The "nipple" projects ·6 cm. above the general inner surface of the cyst first described, and the wall around it, for a radius of about 1 cm., has the characters of ordinary skin, and is thicker than the rest so as to rise slightly beyond the general level, as an areola. The "nipple" is lobulated and bears a few hairs.

Description of the cyst in the opposite ovary.

There is here a single cyst, 4 cm. in diameter, projecting from the outer border of the ovary beneath the fimbriated extremity of the tube. From the interior there projects the crown of a small tooth; a short way above is a low, circular elevation, 1·4 cm. in diameter, and from the lower part of this there projects a very distinct nipple-like process. The cyst wall in these situations presents the wrinkled character of ordinary skin, and both the "nipple" and surface around bear hairs. Section shows the presence of a lenticular cyst beneath the middle of the circular elevation noticed, to the presence of which the elevation is chiefly due. This cyst contained a whitish, thick fluid like mucus. In two other situations there are, in the wall of the main cyst, small daughter cysts, each about ·6 cm. in longer diameter.

Microscopical sections, including the "nipple" and adjacent parts, discover that the former is constituted by clusters of sebaceous glands, typical in structure and in full activity of function; several hair-roots also being exposed in the section.

The epidermis of the surrounding part is of the usual structure. There is a well-marked amount of adipose tissue beneath the "nipple" and neighbouring skin in connection with which groups of perfectly-formed sweat-glands occur.

A thin plate of bone, with corpuscles arranged conformably with the cyst wall, lies in the deeper tissue beneath the situation of the same eminence. The small cyst in the subjacent neighbourhood is lined with tall columnar epithelium, and must be regarded as a daughter cyst of the usual kind in the wall of the primary. It is of the same kind as one at least of the other two displayed in the microscopic section. There is no trace of epithelium in the lower of these other two cysts, which has microscopically an irregularly

spreading limit and has every appearance of being due to degeneration of the wall. Here and there are small bundles of involuntary muscle, doubtless pertaining to the hairs.

Remarks.—It will be seen from these details that there is no production of mammary-gland tissue in this instance. And were it not that Mr. Sutton has traced the stages between such a case as the present and those where mammary tissue is found, the specimen would hardly deserve recording, Mr. Sutton's specimens, however, would indicate that the first evidence of mammary production is a localised hypertrophy of ordinary sebaceous glands, the process giving place in other cases to the modification of such glands, which normally obtains in the development of the breast. Without such evidence it would be difficult to regard the projections in the present specimen as more than a localised hypertrophy of cutaneous structures similar to the well-known condition which forms "lipoma" of the nose.

It would be interesting, in the face of such facts, to determine from what source these cysts derive their nerve supply—secreting, muscular, trophic, and (?) sensory; whether from the nerves of the ovary itself, or whether any new epiblastic formation of nerve tissue arises in connection with an involution from the epithelial elements concerned in the genesis of the cyst.

May 15*th*, 1888.

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6. *A case of congenital syphilis causing lardaceous disease of the viscera, gummata, fibrosis of the liver, and chronic peritonitis.*

By W. Hale White, M.D.

Mary S—, aged 7, was admitted into Guy's Hospital June 8th, 1887. There was a distinct history of syphilis in both parents, who say they have lost four children from croup, one from jaundice at the age of six weeks, and one was born dead.

The patient was a small baby, and at five weeks old had a rash on the privates, cheek, and side. She first complained of pain seven weeks ago; her abdomen began to enlarge six weeks since,

and lately the ankles have become swollen. On admission, the child was seen to be sallow, thin, and weak. The upper central incisors are pegged and slightly notched. The edge of the liver can be easily felt just above the iliac crest; it is hard and has a rounded edge. The spleen may also be felt to extend below the umbilicus; it has a rounded edge. The abdomen is somewhat distended but contains no fluid. The glands in the right axilla are enlarged. There is a considerable painful swelling over the head of the right tibia. There are slight signs of bronchitis, and a basic hæmic murmur may be heard. The left eye shows disseminated choroiditis with some optic neuritis. The urine contains a good deal of albumen, has a specific gravity of 1010, and is of a clear light colour. No other signs of syphilis are present save those mentioned.

At first the child was treated with grey powder, but as even small doses quickly salivated her, mercurial ointment was applied to the abdomen and the node on the tibia. This quickly disappeared, but she did not seem to get on, the liver and spleen becoming very tender to the touch and diarrhœa coming on. She was then ordered Pot. Iod. gr. v, Liq. Arsenicalis ℥iij, and Tinct. Guiac. Ammon. ℥x, made into a mixture and taken thrice daily. By careful feeding, maltine, and this medicine she slowly began to improve, the diarrhœa stopped, some swelling of the feet which was present on admission, the bronchitis, and the abdominal pain disappeared, and she was able to get up. Unfortunately on July 30th she developed a sharp attack of tonsillitis, against which she was quite unable to fight, and she died on August 1st. The albumen was present during the whole of the child's stay in the hospital.

Post-mortem examination.—The liver weighed 61 oz. It was very hard and rounded in shape. The diaphragm was adherent to it. There were radiating fibrous depressions scattered over both surfaces. Section showed that these sent fibrous bands into the interior of the liver. There appeared also to be an increase of fibrous tissue throughout the organ apart from these bands. Just under the upper surface, near the middle of the anterior edge, was a yellow gummatous mass about the size of a small walnut. The organ was lardaceous throughout.

The spleen weighed 16 oz.; it was somewhat hardened, rounded, and very lardaceous.

The kidneys weighed 12½ oz., they were pale, there was a good deal of tubal nephritis, and they were considerably lardaceous. The small intestine was slightly lardaceous. There were several delicate bands of fibrous tissue connecting together the coils of intestine and the omentum to the abdominal wall and the diaphragm to the liver. There was no recent peritonitis or fluid within the abdominal cavity. All the other viscera were normal. The periosteum over the inside of the head of the right tibia was thickened. There was no sign of any recent or old pus-discharging cavity.

Microscopical examination confirmed the points observed with the naked eye. Gentian violet showed an abundant deposit of lardaceous material in the liver, spleen, and kidneys in the usual positions, and a small amount in the cervical lymphatic glands. The sections also showed that there was cirrhosis of the liver, indistinguishable from ordinary cirrhosis, in addition to the gumma and the fibrous bands radiating from the depressions of the surface.

I have brought this case before the members of the Society because it shows the following extremely rare effects of congenital syphilis:

Firstly: Lardaceous disease of the liver, spleen, kidneys, and intestine.

Secondly: Chronic peritonitis.

Thirdly: Fibrous scars and gummata in the liver in addition to the uniform fibrous infiltration. I believe this to be a unique example of lardaceous disease as a result of congenital syphilis. The presence of a large liver and spleen together with albuminuria enabled us to make the diagnosis of lardaceous disease during life.

November 15th, 1887.

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7. *Tubercular tumour of breast.* (*Card specimen.*)

By R. G. Hebb, M.D.

Was removed by Mr. Macnamara from a patient, aged 39, who had had eight children, five of whom are stated to have died of "tuberculosis." The patient had been under treatment for phthisis for many years and had frequently suffered from

hæmoptysis. Six or seven weeks before admission to the Westminster Hospital (November 9th, 1887) she felt a dragging pain in the left breast, and about November 1st, noticed a tumour, which "is a hard nodule about the size of a Spanish nut in the upper and outer part, and in the substance of the left mamma. It is close to the nipple, which is much retracted, and extends upwards and outwards for one and a half inches, and is about three quarters of an inch in width; it is freely movable; the tumour is painful and tender on manipulation. There are a few indurated cords extending towards the anterior fold of the axilla. No enlarged axillary glands."

The growth was removed November 16th, and the patient discharged well November 27th. There was no recurrence when seen two months ago.

Microscopical examination shows that the tumour consists of tubercular nodules, some of which are breaking down in the centre. In one or two places the tuberculosis seems to have primarily involved the ducts, but in most cases the nodules are just without the glandular tissue and embedded within the connective-tissue elements. There are well-marked giant-cells. Several attempts to detect tubercle bacilli failed.

May 15*th*, 1888.

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8. *Case of pulpy growth; tubercle ? in sheath of flexor tendon of index finger; excision with complete recovery; pulpy disease ? of palmar ganglion of opposite hand.*

By Charters J. Symonds.

Martha M—, aged 18, was admitted into Guy's February 26th, 1883, complaining of her right wrist. It was swollen in front, and weak, but this did not prevent her using it for sewing. There was a soft swelling under the flexor tendons, giving the signs and symptoms of a ganglion, and this I thought it to be. It was noted, however, that the swelling did not extend as usual into the palm. She could bear full manipulation of the wrist. Distinct fluctuation seemed to exist. Over the palmar surface of

the head of the metacarpal bone of the left index finger was a rounded, tense, painless swelling, connected evidently with the sheath of the flexor tendon, and giving all the signs of a ganglion. The finger could be bent, but its usefulness was impaired.

On February 27th she was put under chloroform, with a view of having the "ganglion" on the right side opened and drained. An incision under antiseptic precautions was made, and though a synovial sac was opened no fluid escaped, nor was the swelling diminished in size. I could not make out satisfactorily the cause of the swelling, but was inclined to think it a diffused growth amongst the tendons. The almost complete absence of pain appeared to exclude disease of the wrist-joint. A drainage-tube was introduced, and the wound dressed with gauze. Firm pressure was maintained. There was before the operation a good deal of stiffness of the fingers, and later some pain on movement.

March 16th.—Under chloroform an incision was made over the swelling in index finger. It was incised, and instead of the ordinary fluid of a ganglion escaping, a solid gelatinous growth was exposed. This lay *inside* the sheath and was attached to its inner surface. A portion of the sheath, with the growth, was removed. As its nature was doubtful, only a small incision was made into the sheath so as not to interfere with the nutrition of the tendon more than possible. The radial and ulnar arteries were compressed, and the pressure was maintained till the wound was closed and pressure made by dressings and splint on the wound. By the 30th the union was sound and she could move the finger with ease. For a few days there was some reactionary swelling, but no pus ever formed.

The result of the operation may be summarised as follows:

April 9th.—Left forefinger: Movement much improved, in fact is almost complete, whereas before operation it was limited. The sensation is improved. There is no return of the swelling.

The right wrist is not in a satisfactory state. It still swells if the pressure be removed. Movement, especially rotation, is painful, and the hand inclines to the ulnar side. There is probably some disease of the wrist and radio-ulnar joint. There was on this side also a large bicipital gland, an association which led me to suspect growth. She was for some weeks under Liq. Hyd. Per., and was discharged still taking this remedy with iron.

I saw this patient more than a year later, when she had complete

use of her forefinger. The movements of the wrist were impaired but there was no pain and no swelling.

The mass removed from the index finger was about a quarter of an inch in diameter, of a consistence and colour exactly resembling a soft sarcoma. It proved on microscopic examination to be a beautiful example of granulation tissue containing multinucleated cells, exactly resembling the "pulpy tissue" of joints or tubercle as seen in the lung. One may assume that the affection of the right wrist was of the same nature, and that, looking to the great improvement obtained in the movement of the joint, the disease was in the bursa primarily and chiefly. The case seems of interest as affording an example of a swelling in the flexor tendon of a distinctly tuberculous nature, and one that resembled in its clinical characters an ordinary ganglion. The early removal saved the almost certain destruction of the tendon. *January 3rd*, 1888.

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9. *Two examples of hammer-toe.* (*Card specimen.*)

By Samuel G. Shattock.

[With Plate XXXVIII, fig. 3.]

Each of them is a second toe, and in each a careful dissection shows the same condition which I recorded in vol. xxxviii of the Society's 'Transactions,' 1887. The first interphalangeal joint is flexed to a right angle; the second is in a position of simple extension. The flexor tendons are quite normal, the bent position being maintained solely by the lower or posterior portion of the lateral ligaments inserted into the base of the second phalanx. One of the specimens was from a dissecting-room subject, the other from a boy whose leg was amputated by Mr. R. W. Parker for ununited fracture and in whom the opposite foot was normal.

Little holds the malposition to be neuro-muscular in origin, and probably correctly. Of malpositions in the upper limb it more resembles that of the fingers in progressive muscular atrophy than the ordinary Dupuytren's contraction; and it not unlikely depends on some affection leading to loss of power in the interossei. I have at present a foot in which the second toe has the characteristic

position, the first phalanx slightly over-extended, the second flexed at a right angle, and the third in direct line with the second, the extremity of the toe resting on the ground between the roots of the first and third. And in this I intend to examine closely into the condition of the interossei and their supplying nerves. The affection is, in nearly all cases, limited to the second toe, and this was probably so in the example reported in the preceding volume of the Society's 'Transactions.' *May* 1*st*, 1888.

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10. *The stump of a Chopart's amputation.* (*Card specimen.*)

By W. G. Spencer, M.S.

An army pensioner and storekeeper, aged 55, was knocked down on the railway in South Africa on September 9th, 1885. His left foot was badly crushed, his chest was injured, and his right hip dislocated. A primary Chopart's amputation was done at Durban Hospital by Dr. Addison. He was in hospital between three and four months, suffering also from his chest. The amputation wound was not healed on his discharge, and subsequently two pieces of bone came out. He could never put his foot to the ground but walked on a pin-leg. The pin wore down, and that caused such pain in the right hip, which had been dislocated, that he could scarcely walk at all. On admission to Westminster Hospital the remains of the left foot were almost in a line with the leg, the heel very much drawn up. The scar was closely attached to the bone, and the least handling caused intense pain. A Syme's amputation was done on August 31st, 1887, and he can now bear well on his present stump, and is commencing to walk on it.

The specimen shows the scar directed almost vertically downwards, the adhesion of the scar to the astragalus and os calcis, the drawing of the posterior end of the os calcis upwards, the astragalus forwards and downwards, the loss of substance of the anterior portion of the os calcis, and the partial ankylosis between the anterior portion of the os calcis and astragalus.

November 1*st*, 1888.

11. *Fatty herniæ in linea alba.*

By J. Hutchinson, jun.

This specimen was exhibited, not on account of its rarity, for such protrusions of fat in this position are fairly common, but as being a good example of a condition which has led to many errors in diagnosis. The subject from which it was obtained was a well-developed man with his adipose tissue generally present in considerable quantity. Through the skin I could readily detect a tumour in the linea alba, about 5″ from the umbilicus, which was about as large as a small apple. It was irreducible, fairly firm, and somewhat lobulated, and the diagnosis of fatty hernia could be made with some confidence. At the umbilicus itself was a similar but smaller tumour, and here the question of an omental hernia arose. On dissection it was seen that both tumours were outgrowths of subperitoneal fat which had penetrated the aponeurosis of the abdominal muscles and lay in the subcutaneous adipose tissue. From this they were perfectly well defined owing to the difference in colour and lobulation of the two, the fatty hernia being composed of much larger and pinky-yellow fat than the other. The peritoneum opposite to each was puckered and showed small processes in the centre of each, extending for about an inch forwards. These small cavities, or "potential hernial sacs," were quite empty, but it is evident that they might have led to the formation of a true hernia. The same tendency to draw tubes of peritoneum along with the fatty hernia was shown in a series of specimens exhibited by me at the Pathological Society two years ago, obtained from the femoral and inguinal regions. The apertures in the aponeurosis were quite small, the fatty herniæ expanding greatly as soon as they had traversed this layer.

An interesting feature of these tumours, and one which partly accounts for the extreme difficulty with which they are diagnosed from true omental herniæ, is the occurrence of abdominal pain and even vomiting which has been noticed in several cases. Professors Lücke, König, and Kussmaul have drawn attention to this fact, and the two former briefly refer to several cases in which the symptoms were relieved by operative removal of the fatty herniæ (see 'Centralblatt für Chirurgie,' No. 4, 1887, p. 68).

Looking at the traction and compression exerted on the peritoneum, which is drawn after the fatty herniæ in their progress, it is easy to explain such symptoms by irritation of the nerves supplying the former.

Fatty herniæ in the linea alba are most common in its upper part; this, no doubt, is accounted for by the close apposition of the recti muscles just above the pubis. It may well be doubted whether most, if not all, of the supposed cases of omental hernia in the linea alba away from the umbilicus are not really outgrowths of subperitoneal fat, of course excluding the ventral herniæ which sometimes follow abdominal incisions, as for ovariotomy. Most of them are certainly irreducible, as in the present case. Even if operated on, unless the surgeon is fully aware of the frequent occurrence of these fatty herniæ, great difficulty may be found in distinguishing them from omental herniæ. The chief features in the diagnosis are, as pointed out in the case of femoral and inguinal ones (see 'Path. Trans.,' for 1886), the absence of a true peritoneal investment and of any connecting membrane between the lobules of fat.

In a case recently operated on by Mr. Treves at the London Hospital a man had a supposed omental hernia in the linea alba. During the operation it was fully made out that the hernia was simply composed of fat which had come through a small aperture in the abdominal aponeurosis. Such cases are of by no means infrequent occurrence, and their true pathology is hence worthy of more notice than it has received in most English works on surgery. *January 3rd*, 1888.

XII. DISEASES, ETC., OF THE LOWER ANIMALS.

1. *Inguinal herniæ in monkeys.*

By J. Bland Sutton.

Some surgeons are of opinion that a patent funicular pouch is to be regarded as predisposing to the occurrence of a hernia.

It is, however, a remarkable fact that, among mammals, obliteration of this process above the testis is altogether exceptional, indeed, in the majority this process not only remains open but freely communicates with the peritoneal cavity, The only exceptions which have come under my notice are the chimpanzee and gorilla. Professor Owen has found the process obliterated in a chimpanzee. In three specimens of this mammal dissected by me the process was patent.

On this question Hunter writes as follows: "In the adults of quadrupeds, the tunica vaginalis testis communicates with the abdomen, but it keeps nearly the same size, between the testis and abdomen, that it had in the fœtus" ('Essays and Observations,' vol. i, p. 188).

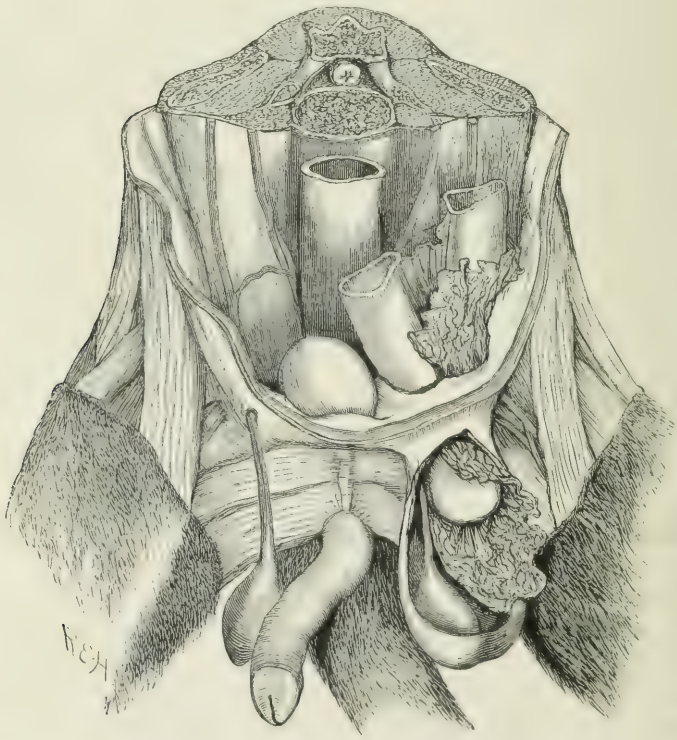
Lawrence, in his work on rupture, states: "In quadrupeds the tunica vaginalis communicates with the abdomen, and yet protrusions of the viscera are rare. In like manner the canal sometimes remains open in the human subject, to even the adult age, without the formation of rupture."

Probably no order of mammals is so interesting as that containing man and the monkeys. Up to the present time I have examined *post mortem* more than 800 monkeys, including examples of all the chief species, excepting the gorilla. In every male, the funicular process was unobliterated and communicated freely with the peritoneal cavity. There was no separation of a piece to form a tunica vaginalis.

Theoretically, if an unobliterated funicular pouch predisposes to hernia, this accident should occur in monkeys frequently. So far I have only seen three cases. In each instance they were of the variety known as congenital, the contents completely surrounding the testis. Two were epiploceles. One was found in a round-faced monkey, *Macacus cyclopis*; it occupied the left funicular process. The second occupied the process in a specimen of *Macacus sinicus*, the Bonnet monkey, and is described and figured in the 'Proc. Zool. Soc.,' 1886. The first specimen is preserved in the Museum of the Royal College of Surgeons.

My third specimen is an entero-epiplocele of the congenital variety, lodged in the left pouch of a Rhesus monkey, *Macacus*

Woodcut 27.



The pelvis of a monkey. The left funicular pouch is occupied by a piece of small gut and omentum.

rhesus. As in the preceding monkeys there were no adhesions. The parts are represented of nearly natural size in Woodcut 27.

The Museum of the Royal College of Surgeons contains a Hunterian specimen of this defect. It is described as "a very large inguinal hernia from a monkey." The sac contains a portion of colon, with omentum, both in a healthy state. The testicle is below the sac.

As it was impossible to make out accurately the relation of the hernia to the testis, I obtained permission to cut the specimen down and make a closer inspection. To my surprise the following condition was found :—

The contents of the sac were a large loose layer of delicate omentum, in the folds of which some six or seven hydatids were lodged. These cysts are of the dimensions of large gooseberries but quite round; one of the cysts is loose in the hernial sac. About four inches of colon dipped into the pouch. This specimen is of interest, as here for the first time in any mammal other than man, I saw a testis with a distinct tunica vaginalis. It is unfortunate that no other details concerning this specimen are forthcoming, not even the name of the monkey to which it belonged. The parts are shown nearly the size of nature in Woodcut 28. With the exception of Hunter's specimen and the three observed by me, I know of no other specimen of hernia in monkeys.

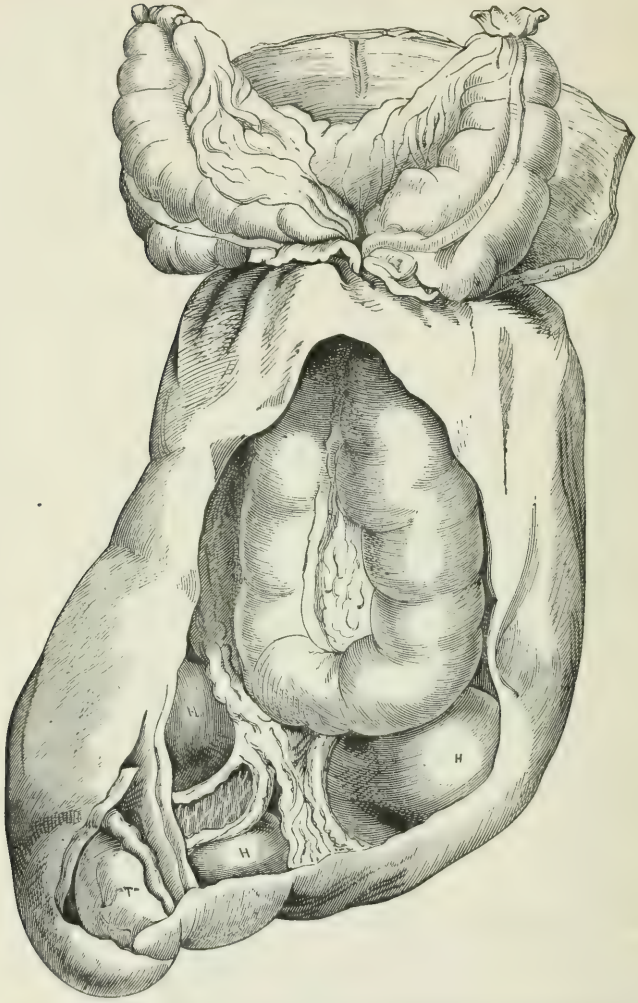
Lawrence mentions that Wrisberg saw a scrotal hernia in a horse, and observes that monkeys have been affected in the same way. This, however, goes for little.

It perhaps should be mentioned that the existence of hydatid cysts in Hunter's case is capable of easy explanation. Hydatids are very common in the peritoneal cavity of the monkey, free from any attachment; hence if the sac of the hernia presented a large neck and these cysts were present, they would easily find their way into the pouch by gravitation.

It is necessary to avoid mistaking lipomatous masses in the spermatic cord for hernial protrusions. These simulate epiplocels as closely in mammals as in the human subject, and in their lobulation resemble omentum. They occur in some of the rodents, such as the Gambia pouched-rat, the marmot, in the vulpine phalanger and opossum.

Herniæ occur with a fair amount of frequency in domestic mammals, sheep, goats, horses, pigs and the like, but as yet I

Woodcut 28.



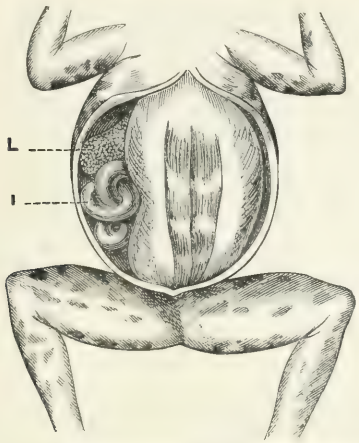
The sac of an inguinal hernia from a monkey, containing a piece of colon, omentum, and hydatid cysts H. T. The testis.

have never seen an inguinal hernia in a wild mammal except the monkey.

Perhaps one may mention as a curiosity the following case which

occurred in a frog. Whilst examining a number of frogs for another purpose, I accidentally found, on opening the abdominal lymph-space, that a coil of intestine and the posterior extremity of

Woodcut 29.



A frog with a portion of intestines and the posterior extremity of the right lung occupying a lymph-space.

the right lung had become herniated through an old rent in the abdominal muscles, into the large lymph-space which is so conspicuous a feature in the anatomy of this vertebrate.

November 15*th*, 1887.

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2. *Multiple intussusception in a baboon.* (*Card specimen.*)

By Walter K. Sibley, M.B.

The specimen is from a female South African baboon which had lived in the gardens of the Zoological Society for about two years. The larger portion of the small intestine is intussuscepted, only thirty-seven inches not involved, and this portion is apparently healthy. The main lesion is at the lower end of the ileum, some

feet of which are displaced through the ileo-cæcal valve, and lie in a spiral manner and in a necrotic condition in the cæcum. Just above this the walls of the gut are thin and discoloured. Some four inches higher up a second apparently more recent invagination has occurred, and another less marked one just below this. Here the gut has in the process undergone some twisting.

There were no adhesions or fluid in the abdominal cavity, the mesentery was much congested, and its glands enlarged. The small intestine was nearly empty, the large being filled with hard fæces and a few nematodes. The other viscera not obviously diseased.

The clinical history revealed menorrhagia for some months, and an acute illness of about a week's duration.

February 7th, 1888.

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3. *Sarcoma of palate of an otter hound.* (*Card specimen.*)

By H. A. Lediard, M.D.

From a bitch from the Carlisle pack of otter hounds, aged 8 years. The tumour was not apparently the cause of pain or difficulty in swallowing. The duration of the growth is uncertain, and the dog was destroyed as unfit for breeding purposes.

From the soft palate on the right side a tumour the size of a large walnut springs so as to project downwards towards the base of the tongue. It is unconnected with bone and has a broad attachment.

Microscopic examination shows small, round, and spindle-shaped cells in great number without any definite stroma.

October 18th, 1887.

4. *Fracture of the larger pastern bone of a horse.* (*Card specimen.*)

By William Collier, M.D.

The horse whose fractured pastern bone is shown was seen a few weeks since trotting along the road in a light gig, when suddenly it stopped dead lame, and was with difficulty walked to some stables about thirty yards farther on. On examining the fetlock-joint of the near foreleg distinct crepitus was obtained. I expressed the opinion that the bone was fractured. Two days later, by the advice of a veterinary surgeon, the horse was killed. The horse was blind, and consequently had very high action. Fracture of this bone occasionally occurs in racehorses when galloping at full speed on hard ground, also among hunters as the result of jumping, but it would seem almost impossible for a horse trotting along the road with no weight on his back to put his feet down with sufficient violence to produce so extensive a fracture. The horse was in regular work, and quite sound as far as could be judged. *May 15th*, 1888.

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5. *Abscess of brain, with tumour of the pituitary body and abscesses in the lungs, in an ewe.*

By Walter K. Sibley, M.B.

The history of the case, which was an ewe, aged 2 years, is briefly the following:

About the middle of last August it appeared generally ailing with loss of appetite; it would become separated from the rest of the flock, and remain in an apathetic condition for hours. In time it seemed to be blind—for it would run into a gate or even a dog—and subsequently deaf. Also it had difficulty and straining at micturition, but with no obvious paralysis.

Becoming more feeble and completely apathetic, it was killed on September 30th.

Post-mortem.—General condition good, much fat present in the abdomen. Bladder contained about one and a half pints of clear urine. No obvious lesion in the other abdominal viscera. There were considerable adhesions about the left pleura, and both lungs contained a number of rather firm nodules of consolidation and some soft ones containing inspissated pus, the nodules varying in size from a pea to a small bean, being more numerous in the left lung. In places the smaller bronchioles were dilated and filled with débris.

The whole brain was much distorted, the right half appearing much larger than the left, together with partial obliteration of its convolutions. The membranes were generally inflamed and thickened, and in the right parieto-occipital region a large abscess was present, most extensive on the upper surface and involving the whole depth of the cerebrum in this region to the base of the skull. The abscess, which was markedly circumscribed, contained thick creamy pus and extended through the occipital and temporo-sphenoidal lobes of this side.

There was considerable roughness of the inner surface of the cranium, especially in the left occipital and right frontal regions, also on both orbital plates of the sphenoid more marked on the left side, and in the median plane of the base of the skull from the olivary process to the foramen magnum. There was no evidence of disease of the middle ear on either side. The part of the base of the skull for the reception of the pituitary body, which in a sheep is the size of a small bean, was occupied by a swelling as large as a small walnut, and of a deeper colour than the surrounding tissues. The floor and sides, especially the right, of the sella turcica were eroded, the dorsum sellæ and posterior clinoid processes being completely absorbed; the body of the sphenoid was very thin and almost perforated into the pharynx. The dura mater around was thickened and firmly adherent to the posterior border of the growth, appearing to completely enclose the mass separating it, except at the attachment of the infundibulum from the surface of the brain above, and from the base of the skull below, the tumour lying below the circle of Willis and its included parts, and the anterior part of the pons, having on either side the crura cerebri, being partially enclosed by the

temporo-sphenoidal lobes. It presented anteriorly a bilobed appearance, the right side being larger than the left, the groove between the lobes extending for some distance into the substance of the body, the whole being more or less heart-shaped, with an apex anteriorly and a base posteriorly.

Microscopical examination of the lungs showed them to be studded with groups or worm-knots of nematodes (*Pseudalius ovis pulmonis*), each group consisting of a collection of larvæ in various stages of development; occasionally an adult worm was found in section, often with large numbers of eggs in its neighbourhood. The older nodules present some necrotic tissue in the centre, surrounded by a zone of large epithelioid cells, and this again by small cells, with very frequently some small-celled infiltration of the lung tissue around.[1] Section through one of the abscess cavities showed its walls to be formed of a dense collection of fibrous tissue and small cells. The débris in the bronchioles was chiefly composed of highly organised vegetable tissue containing spiral bundles, an occasional nematode being present among these.

Section of the enlarged pituitary body showed it to be composed of regular, large, more or less cubical, epithelial cells, each with a large granular nucleus, one group of cells separated from the next by strands of fibrous tissue, thus arranged in alveoli, with all the appearances of an adenoma. In places are roundish spaces, surrounded with a capsule of fibrous tissue, lined by small cubical cells, and containing a colloid material and other metamorphosed tissues; also some smaller spaces filled with fibrous tissue and débris. At one part of the section the structure changes slightly, the cells become more irregular in size and arrangement, and have a larger quantity of fibrous tissue mixed with them. A few blood-vessels are present.

It might perhaps naturally suggest itself that the abscesses of the lungs were caused by the parasites, and that the abscess of the brain was of a pyæmic nature produced by septic emboli from the lungs, as is considered to occur in the human subject. But entozoa in the lungs of lower animals are of very common occurrence, and beyond setting up some degree of local inflammation, and then becoming encysted, do not as a rule cause further trouble. In some districts and in certain seasons nematodes may be found in

[1] *Vide* H. Bewley, 'Journal of Anatomy and Physiology,' 1887.

the lungs of almost every sheep examined, in most cases apparently quite healthy subjects, in which their presence has not been suspected during life.

On the other hand, it might be maintained that from an abscess of the brain pyæmic abscesses of the lungs had been produced.

Still another supposition is possible, namely, that the cerebral abscess had been set up by an affection of the cranial bones, and that this morbid condition of the central nervous system, disturbing the normal correlation of nerve influence between the brain and the pulmonary tissues, their nutritive changes, essential to functional activity, are interfered with. In other words, the vitality of the lung tissue, which in this particular case was already damaged by the patches of parasitic pneumonia, becomes lowered. So as the result of this trophic change the nodules of consolidation produced by the parasites break down and form abscesses, these not being the result of pyæmia, that is of septic emboli, but of direct nervous nature. In fact they might be placed in the class of the trophoneuroses. As appears to be frequently the case, the lung on the opposite side of the body to the primary nerve lesion is the first, and occasionally the only one affected.

The presence of inhaled vegetable tissues in the dilated bronchioles might tend to show that the sensory mechanism of the bronchus and bronchioles was impaired, and so foreign bodies were permitted to lie undisturbed on the mucous membrane.

This theory appears to have some ground to support it from many of the recorded cases of so-called pyæmic abscess of the brain associated with abscess of the lung, for some of the facts mentioned in connection with these cases would apparently indicate that the abscess of the brain was primary, and the abscess of the lung secondary; and that the general symptoms of pyæmia were absent.

Tumours of the pituitary appear to be somewhat rare. Some five are recorded in the 'Transactions' of this Society, namely, "Cancer in a Baboon," by Goodhart (vol. xxxvi); "Myoneuroma," by Hale White (ib.); "A Mixed-tissue Tumour (Connective Tissue, Epithelium, Cysts, and Bone)," by Bowlby (ib.); a case by Taylor (vol. xxxv), and another by Ware, with no histological descriptions. Also Anderson, in 'Brain' (vol. ix), "A Case of Sensory Epilepsy caused by a Vascular Tumour of Pituitary," and a very similar case by Nettleship in the Ophthalmological Society's 'Transac-

tions' (vol. iv, p. 285). Michel, in the 'Med.-Chir. Review' (1862, ii, p. 93), records a case in a negro, but with no histology.

Rokitansky[1] speaks of them as not uncommon; and of three classes: Tuberculous, cancerous, usually of the medullary type; and colloid, growing in the spaces between the lobes.

Ziegler[2] states that cystic degeneration and hyperplastic overgrowth is not uncommon, this condition is called adenoma. Also carcinoma, sarcoma, lipomata, and teratomata occur; tubercles and gumma are rare.

From its position and structure this specimen is probably an example of hyperplastic overgrowth and cystic degeneration or adenoma, affecting chiefly the anterior lobe or that part of the pituitary body developed from the stomodæum.

January 17*th*, 1888.

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6. *Horny tumour in mouse.* (*Card specimen.*)

By Frederick Treves.

An adult mouse with a large horny tumour, nearly one inch in length and a fifth of an inch in width, growing from the dorsal median line of the neck over the upper cervical vertebræ. The mass is horny to the feel and projects directly upwards. It has evidently developed from a dermoid cyst situated between the occipital bone and the upper cervical vertebræ. On section it is shown to be composed of dry sebaceous matter. Such dermoid cysts are not uncommon in mice and not infrequently lead to the formation of horns. A very similar specimen exists in the Museum of the Royal College of Surgeons. *March 6th*, 1888.

[1] 'A Manual of Path. Anatomy' (Syd. Soc. Tr.), vol. iii, p. 433.

[2] 'Special Path. Anatomy' (Macalister's trans.), sect. xi, p. 326.

7. *Tuberculosis in fowls.*

By Walter K. Sibley, M.B.

[With Plate XXXIX.]

The subject of avian tuberculosis is not new to the Society; several cases of tubercular disease in birds were long since recorded by Dr. Edwards Crisp, and more recently a paper by Mr. J. Bland Sutton and Dr. Heneage Gibbes on the same subject appeared in the 'Transactions.' As, however, I have had the opportunity of observing a series of cases in fowls I thought it might be well to bring them before the Society, mentioning, as far as possible, only those points which appear not to have been dwelt upon. For some few years past on a farm in Surrey a disease has appeared among the poultry; several adult birds have wasted and died. The fowls are at first fine and healthy, well developed, and have good plumage. All goes well till they are about two years old, when symptoms appear. The *post-mortem* lesions were in the main the same in the several cases, only differing with the degree to which the disease had progressed, some birds having died from the disease, others were killed in various stages for the purpose of examination.

The abdominal organs, that is, the intestine and liver, are the parts primarily affected. In the intestine small areas of infiltration appear in the submucosa, consisting of a collection of leucocytes with fine strands of fibrous tissue, with here and there a larger cell. The villi over this region soon become infiltrated, at the same time the process extends into the muscular layer, and the serous becomes thickened. The centres of the larger collection of cells soon caseate, the mucous membrane over them gives way, a small ulcer with thickened edges is formed, with at first a very minute aperture into the bowel. The deposits then break down more rapidly than the mucous membrane which thus becomes undermined, and so a sacculated cavity, extending on the outside of the gut and filled with débris is formed. Its walls go on increasing from fresh deposits of tubercle together with thickening of the serous coat. From time to time the débris from the centre of this is discharged into

the lumen of the digestive tract, and thus an enormous quantity of caseated material is expelled from the system. In the above way caseating masses or pouches, varying in size from a split pea or a marble, or even, in one case, to a pigeon's egg, are produced. These masses, though more prominent externally, yet cause considerable mechanical obstruction to the progression of the intestinal contents, so the bowel becomes dilated above each of these, giving the whole digestive tract a very irregular appearance. In those cases in which the cæca were affected, the chief deposit was at their junction with the main gut, and so they became much dilated, especially at their extremities, which in some cases contained entozoa (*Ascaris vesicularis*). Frequently the œsophagus or crop presented lesions having the same characters, and in one case there was a small circular ulcer in the gizzard.

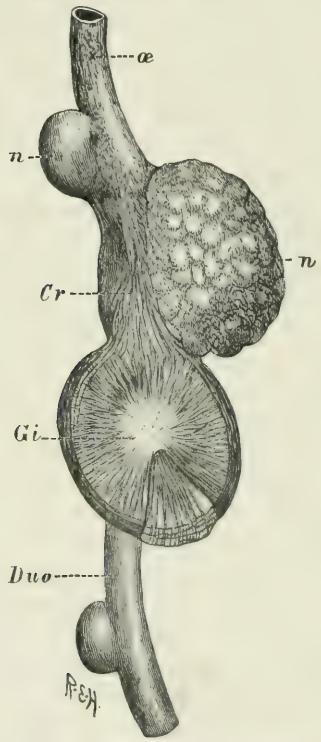
The largest pouch found was from the posterior wall of a crop (which in this case was much atrophied). This pouch was the size of a pigeon's egg, and resembled a sacculated aneurism. It was filled with débris from caseation, and also partially digested food-stuff. Its walls were composed of caseous matter and fibrous tissue; it communicated with the crop by a small, nearly circular opening about half a centimetre in diameter. A second smaller sac, about the size of a marble, was present a little higher up, projecting from the anterior wall of the œsophagus. Under the microscope a section through the wall of this pouch showed it to be composed of an external layer of fibrous tissue and an internal layer of metamorphosed tissue, in places caseous, and becoming more so towards the internal surface, where it appeared to be crumbling away. In the deeper parts of the fibrous tissue were large spaces filled with small cells. There was considerable small-celled infiltration at the junction of this with the metamorphosed tissue, where the fibrous tissue bundles appear swollen and granular. Enormous numbers of bacilli were scattered both in groups and singly towards the internal edge of the degenerated tissue, but few were found in the deeper regions and at the junction with the fibrous tissue, where the process of decay is most active (Plate XXXIX, fig. 3).

In all cases in which the gut was affected the mesentery was more or less infiltrated with small cells, especially along the line of the blood-vessels, and in many places circumscribed areas of leucocytes, in some places completely surrounding and in others on

the side of small capillaries were seen; more advanced stages with caseation were also numerous.

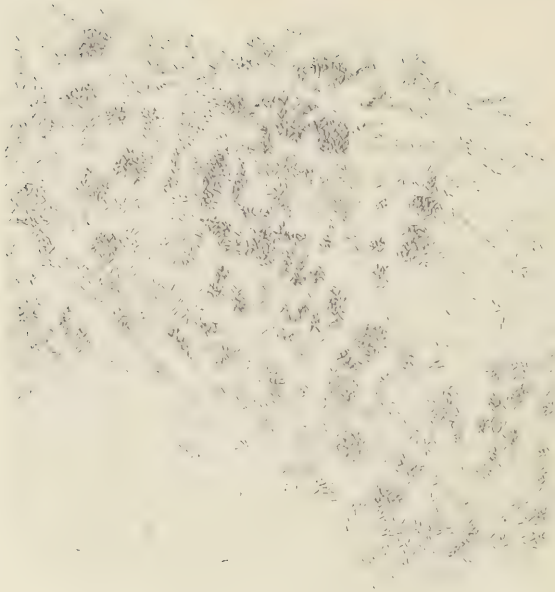
Examination of the liver showed the whole organ to be studded with circumscribed roundish areas in various stages of development;

Woodcut 30.



Showing sacculated pouch from wall of crop (one half nat. size). *œ.* Œsophagus. *Cr.* Crop, atrophied. *Gi.* Gizzard. *Duo.* Duodenum. *n, n.* Nodules.

in the smallest ones nothing but a collection of leucocytes could be detected; in some nodules with one or more larger cells in the centre, also fine strands of fibrous tissue scattered irregularly about, especially at the periphery. No blood-vessels could be seen in them. When slightly larger the cells of the central region disintegrated and became caseous, so that in a more advanced stage each nodule consisted of an irregular central mass of caseation,



DESCRIPTION OF PLATE XXXIX.

To illustrate Mr. W. K. Sibley's paper on Tuberculosis in Fowls. (Page 464.)

FIG. 1.—Section of liver (Zeiss A, oc. 2). Nodules in various stages of growth and degeneration. The central region shows mass of caseation at periphery of which groups of bacilli are seen. This is surrounded by a zone of epithelioid cells and leucocytes. In the upper part a small secondary nodule is seen outside the area of caseation.

FIG. 2.—Portion of above section more highly magnified. (Zeiss D, oc. 2.)

a. Central caseation showing irregular outline of periphery.

b. Group of bacilli.

c. Epithelioid cells.

d. Leucocytes.

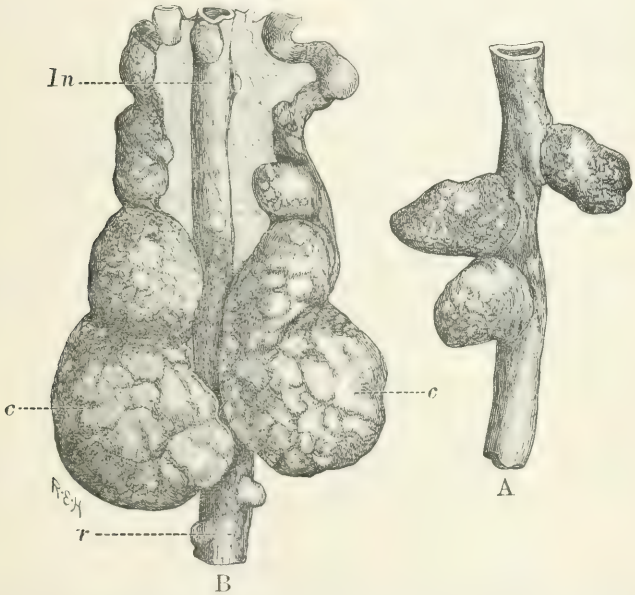
e. Fibrous tissue.

f. Liver-cells.

FIG. 3.—Free margin of wall of caseous pouch growing from the crop, showing bacilli.

extending in the form of pseudopodia into the next area; bacilli were present, usually in groups, towards the periphery of this degenerated tissue (Plate XXXIX, figs. 1, 2). Between these processes of progressive caseation large epithelioid cells were seen together with a few small cells, and then a zone of leucocytes with few wavy bundles of fibrous tissue, more fibrous tissue with fewer cells surrounding and separating the whole from the liver substance; occasionally there was some infiltration of leucocytes among the liver-cells, which themselves often appeared swollen

Woodcut 31.



A. Piece of small intestine with large nodules (one half nat. size). B. Intestine at junction of cæca, showing considerable deposit in wall of latter (one half nat. size). *In.* Intestine. *r.* Rectum. *c c.* Cæca.

and granular in the immediate proximity of the nodules. Many which had caseating centres presented small tubercles in the process of formation at their peripheral regions, these revealing all the characters of the newly-formed nodules described above. As the disease progresses a gradual increase both of deposit and of fibrous tissue takes place throughout the whole organ, the liver-cells

atrophying. At the line of junction of caseation with living tissue there was a similar appearance as that described above in the case of the pouch from the crop.

In most cases the lungs presented no obvious lesion, in a few there were slight pleural adhesions, usually posteriorly, occasionally with fluid in the pleural cavity. The most frequent lesion found was a small isolated nodule the size of a split pea on the anterior surface of the lung towards its base. Upon section it was of a cartilaginous consistence in the substance of the lung, though close to the surface and covered by thickened pleura; in no case was there any deposit at the apex or any cavity formed. In one specimen nearly the whole of one lung and the base of the other were caseating masses, but this appeared secondarily by direct extension from caries of the adjacent vertebræ. Here the process had also extended up the bronchus into the trachea, and up this into the syrinx. The deposit was chiefly in the adenoid tissue posteriorly, and visible upon opening up the trachea as small pale projections beneath the mucous membrane along the posterior border. The process diminished in quantity as one proceeded from below upwards, very abundant and almost surrounding the bronchus of this side; it gradually decreased until the syrinx was reached in which recent deposits only were present, those lower down having undergone caseation.

Microscopically the whole lung was often seen to be more or less infiltrated with small cells, and in some cases circumscribed areas of leucocytes leading to nodules of caseation with thickened pleura over them. A section through one of the more recent nodules showed great infiltration of the peribronchial region with small cells. In one case, in the centre of some of these smaller infiltrated bronchioles, a fungus (*Saccharomyces mycoderma*) was present.

A section from about the middle of the trachea showed much submucous infiltration posteriorly with scattered nodules of caseation containing large numbers of bacilli.

In a few cases there were caseating lymphatic glands in the lower and deeper tissues of the side of the neck, but in none had they suppurated or discharged externally, nor did there appear to be much infiltration of the tissues around. Usually the kidneys and ovary were normal, in a few there was some deposit about the hilus of both, and occasionally the disease had penetrated into the substance of the organ.

In two cases, in both of which the visceral lesions were in an early stage, some caries of the vertebræ was present, and in one also hip-joint disease with an old abscess cavity in the upper part of the thigh-bone.

A case of disease of the spinal column presented in the abdominal cavity a large abscess sac containing some two ounces of flaky fluid, extending on the left side posteriorly down into the pelvis and displacing the spleen and liver. The spleen was raised forward and firmly adherent, being almost embedded in its wall; the left lobe of the liver was also adherent to the anterior surface, and the kidney of this side to the posterior surface. Upon opening up this sac its walls were found to be thin and very friable, covered internally by an irregular deposit of flaky material and containing lymph-like pus with solid bodies suspended in it. This main sac communicated through the rudimentary diaphragm by a narrow passage with a small cavity in the base of the left pleura (the pleura, lung, and diaphragm being one cheesy mass). The whole lung of this side, with the exception of the apex, was solid and adherent to the spine, upon separation from which some caries of the bodies in the dorso-lumbar region was exposed extending over some four or five vertebræ. The abscess appeared to originate from this, pus channels being traced to the diseased bone. There was also some caseation of the base of the opposite lung with fluid in its pleura in connection with the spinal caries, and pus channels going through to the other side.

In another case there was hip-joint disease on the left side, which was chiefly confined to the acetabulum, the floor of which was replaced by fungating masses of granulation tissue projecting into the interior of the pelvis; there was little or no fluid in the joint, but considerable matting together of the tissues in the neighbourhood. The head of the femur was movable and had slight erosion of its cartilage. The thigh-bone readily fractured through its neck, and it was then found that in the region of the tuberosities was a cavity filled with a very dry nodule of caseated material the size of a large pea. The walls of this cavity were quite smooth and apparently of old standing, the caseated nodule being firmly encapsuled by the surrounding bone.

In the region of the lower dorsal spine was slight erosion of the bodies of two adjacent vertebræ, the right pleura being filled with flaky blood-stained pus and in close contact with the

diseased bone. The lung itself was healthy; no fluid in the opposite pleura.

Microscopical section through the neck of the femur showed some of the so-called air-cavities to be enlarged and filled with small cells, some of which in places were undergoing caseation; numerous bacilli were present in these patches of degeneration.

Bacilli, which both structurally and in their reaction to staining fluids, were apparently similar to those of tubercle bacilli found in man, occurred in great numbers in the following regions: liver, spleen, intestine, œsophagus, crop, lung, trachea, lymphatic glands, and femur.

In all of these examples the bacilli were present in a tissue in which apparently (according to their reaction with staining fluids) some process of degeneration had commenced. Although in great abundance in these regions they were not found in cells which had not undergone some degree of metamorphosis. From this it would appear that the bacilli were the result and not the cause of the disease, and that they were produced and grew in the products of degeneration; they soon absorbed the essential elements from the soil in which they grew, and so disappeared from the older regions of caseation and abounded in those of more recent formation. Hence they occur towards the periphery of the caseous nodules in the viscera and towards the free edge of the degenerating tissue in the walls of the pouch from the crop, and not in the deeper layers where the process of gradual metamorphosis of living cells into a condition of coagulative necrosis is going on.

It would seem that the tissues of the avian tribe offer a soil in which tubercle develops to a much more extensive degree than in the human subject, and that the life-energy of a bird is greater, withstanding a more prolonged strain upon its molecular constitution before the dissolution of the individual. So the collection of cells very rapidly degenerates, the caseated products becoming very irregular in outline, with bacilli present in great numbers, usually in groups towards their periphery. Structurally the masses of caseation were very firm; in some cases a small furrow had appeared around them; they tended to become separated from the tissue in which they were formed, as seen on the surface of the liver. In some birds (pheasants) I have found these small caseated nodules formed in the liver lying free in the abdominal cavity and others on the surface of the liver in the process of separation. The

deposits appear never to go beyond the stage of early caseation; do not calcify or of themselves break down and form cavities. There is a great tendency to spread by continuity, as in the instance where the disease spread from the spine to the pleura, lung and bronchus and then up the trachea to the syrinx. Not only is caseation very extensive but there is frequently considerable tissue formation previous to complete degeneration, so the nodules were large compared to the size of the animals: in fact the disease closely approached a neoplasm in its nature.

The above cases are very interesting from an etiological point, the disease being unknown in the other poultry yards in the neighbourhood, many people about stating that they had never lost an adult bird from disease. The hygienic conditions of the locality are very good, being on the south side of chalk downs, fowls free in a large field, huts water-tight and well ventilated. The tubercular disease in these cases appears to have been limited to certain broods; others which had lived for some years along with these remained exempt from the affection.

The disease is a clear case of hereditary transmission, and must have been brought into the farmyard some few years ago when the stock was first started, and have propagated itself with fatal regularity, in spite of every attempt to eradicate it by killing off and carefully getting rid of all affected birds. Here hygiene and good feeding have done nothing either to check the outbreaks of disease or to stop it when it has once appeared in any given case. The only plan appears to be to kill off the whole stock and start afresh, for though frequently for some months no illness occurs, then, as soon as the birds get to a certain age, they one by one, sicken, emaciate, and die. *December 6th*, 1887.

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8. *Tuberculosis of fowls.* (*Card specimen.*)

R. G. Hebb, M.D.

The two livers exhibited were taken from fowls bred in Scotland, near Glasgow. The owner stated that the birds were dying of some wasting disease, and that the liver was the part most affected.

Two specimens were sent to me: one was large, soft, very friable, and the seat of numerous spherical yellowish masses, varying in size from a hemp-seed to a Spanish nut. These masses were enclosed in a thin capsule which sharply separated the contents from the adjacent hepatic tissue. Microscopical examination showed that these degeneration-masses, which were quite devoid of any definite structure, were filled with bacilli. These bacilli gave the same colour reaction as those of tubercle bacilli.

The second liver was apparently quite healthy to the naked eye. Indeed, the bird was served at table, the liver being sent merely for comparison. Microscopical examination, however, showed numerous tubercles in various stages of growth: (1) A collection of small lymphoid indifferent cells. (2) Collections of cells, partly lymphoid and partly epithelioid in character, surrounded by a definite capsule consisting of fusiform fibres. (3) Tubercles with central caseation and surrounded by a fibrous capsule.

In this liver no micro-organisms could be demonstrated, although the sections were submitted to exactly the same test as those of the first case.

It is obvious that the second bird had suffered and recovered from the tuberculosis, but the inference therefrom is doubtful. That is to say, had these last tubercles become obsolete, or was the disease still capable of future development? or in other words, is this condition to be regarded in the light of a "vaccination," or to be considered as merely quiescent? *December 6th*, 1887.

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9. *On an exostosis from a fish.*

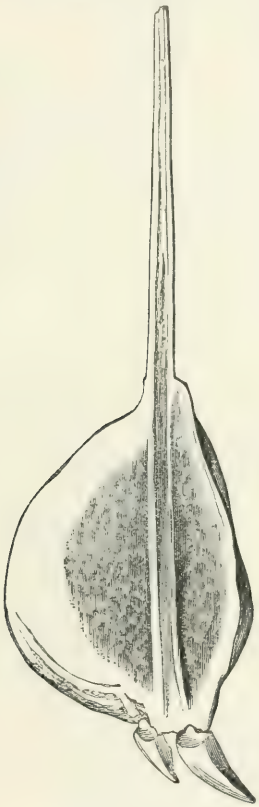
By J. Bland Sutton.

A few weeks ago Dr. Rundle, of Truro, sent me a specimen which he described as resembling an ossified bird's head. He was quite sure it was not artificial as the source from whence he received it was above suspicion. There was no difficulty in determining its nature. The specimen in question is an osteoma connected with the fin-ray of a fish, probably *Ephippus*.

It occurred to me that it might be of some interest to bring

under the notice of the Society a few facts relating to these singular tumours, with the hope that some other examples may be secured which will enable us to come to some conclusions as to their nature.

Woodcut 32.



A fin-ray of Ephippus, with an osteoma, seen in section.

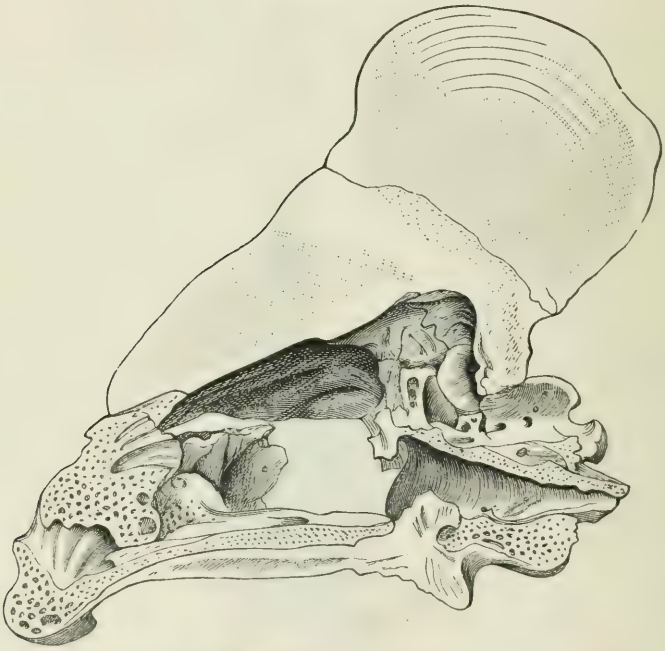
As far as I can learn, the first description of these tumours was published by William Bell[1] in 1793. Bell described his specimen, a *Chætodon*, as being about eighteen inches long, thirteen broad, and

[1] 'Phil. Trans.,' 1793. Bell was at one time assistant to John Hunter.

about three in thickness. It is frequently caught at Bencoolen and several other parts of the West Coast of Sumatra.

The skeleton is very singular, many of the bones having tumours, which in the fish Mr. Bell saw, he supposed to be exostoses arising from disease, but on dissecting a second specimen found the bones had exactly similar tumours, and the fishermen informed him that

Woodcut 33.

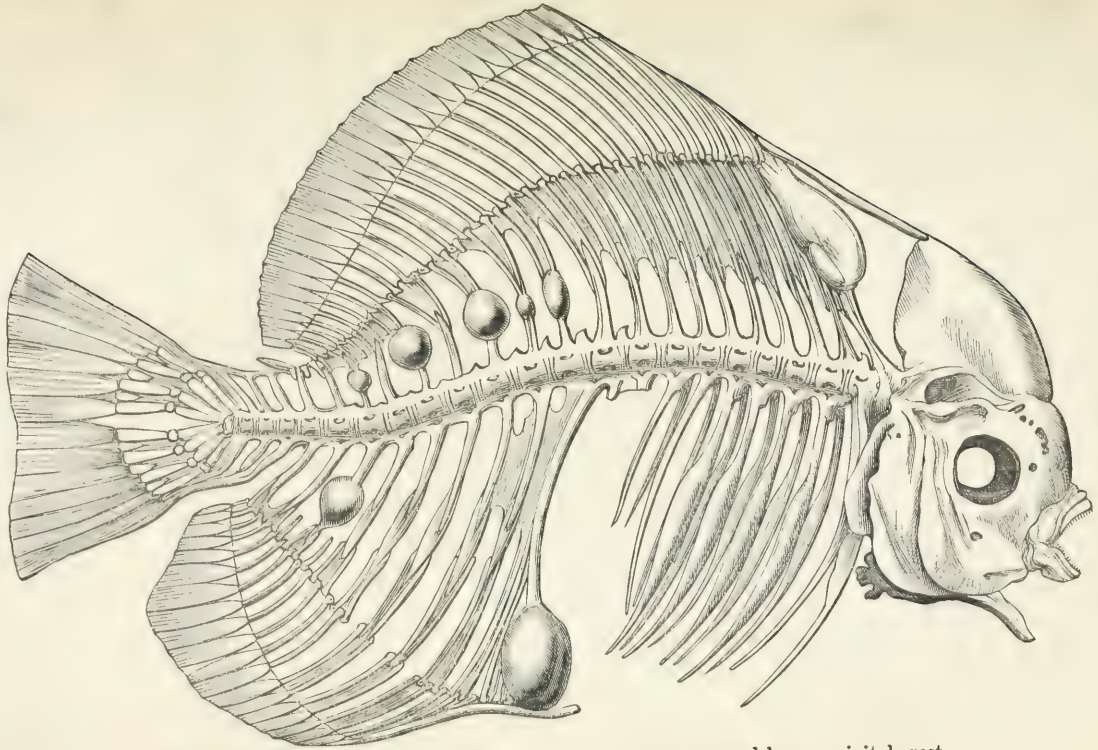


Section through the skull of *Ephippus gigas* showing a large osteoma surmounting the occipital crest.

they were always found on this fish; he therefore concluded them to be natural to it. The tumours were soft and spongy as to be easily cut with a knife; they were filled with oil.

Bell was well aware that Hunter's collection contained two or three of these bones, but he never knew to what fish they belonged. They were supposed to be from the backs of some of the larger Rays.

Woodcut 34.



Bell's specimen of *Chætodon arthriticus* with its bony tumours and large occipital crest.

The Museum of the Royal College of Surgeons contains nearly a score of these bones; they are all described in the catalogue by Professor Owen as belonging to the light-horseman fish *Ephippus* and labelled *Hunterian*. These are probably the specimens referred to by Bell as being in "Mr. Hunter's collection." The rays with their tumours are the exact counterpart of those figured by Cuvier and Valenciennes[1] as common to the anal fin of *Ephippus*. These rays are very characteristic, for they present at one end a tumour of the size of a chestnut, hard, smooth, and as dense as ivory; articulating with this by means of a shackle-joint is one, but usually two, smaller rays. On section the outline of the ray can be clearly defined running through the midst of the tumour. A typical specimen is shown in section (Woodcut 32).

Cuvier was of opinion that these exostosed rays come from the anal fin of *Platax noduleux*, and that Bell's specimen is really a *Platax* and not *Chætodon arthriticus*. Cuvier further states that these bones are common in osteological collections. They are brought home by travellers who have eaten these fish, and preserved their fin-rays as curiosities.

Closely allied to these tumours of the fin-rays are the very remarkable bony masses which Valenciennes has described in connection with the skeleton of *Ephippus faber* and *Ephippus gigas*. In the former fish they affect especially the occipital crest. In old fish it forms a large boss of bone the exterior of which presents an appearance like enamel. (Woodcut 33.)

As far as I know, no explanation has been offered to account for these tumours.

As the sketch illustrating Bell's original description of *Chætodon* is very rough and inadequate, and as the skeleton is preserved in the Museum of the Royal College of Surgeons, I have had it redrawn (Woodcut 34). *March 20th*, 1888.

[1] 'Histoire Naturelle des Poissons,' t. vii, p. 204.

ADDITIONAL LIST OF CARD SPECIMENS EXHIBITED DURING THE SESSION.

1. Hæmatoma of dura mater. By Angel Money, M.D. November 1, 1887.
2. Sarcoma of dura mater (recent specimen). By Angel Money, M.D. March 20, 1888.

3. Interlobular emphysema of the lung, due to pressure of an aneurysm of the thoracic aorta on the trachea (with drawing). By G. N. Pitt, M.D. May 15, 1888.

4. Aneurysm of the heart. By F. G. Penrose, M.D. May 15, 1888.
5. Ulcerative endocarditis (recent specimen). By F. G. Penrose, M.D. March 20, 1888.
6. Valvular disease of heart in an infant. By B. G. Morison, M.D. April 3, 1888.
7. Five cases of tricuspid disease. By R. G. Hebb, M.D. December 6, 1887.
8. Aortic aneurysm perforating the trachea. By Angel Money, M.D. October 18, 1887.
9. Aortic aneurysm pressing on left bronchus; solid lung with apical excavation; basal empyema. By Angel Money, M.D. October 18, 1887.

10. Two cases of cancer of the œsophagus involving the trachea and necessitating tracheotomy (preserved in University College Museum, 1039[A] and 1039[B]). By C. Stonham. May 1, 1888.
11. Imperforate anus. By G. N. Pitt, M.D. May 1, 1888.

12. Multiple lymphadenomata of stomach and intestine. By G. N. Pitt, M.D. May 1, 1888.
13. Lumbar hernia through an abscess. By E. Owen. January 3, 1888.
14. Gummata of liver in infant (recent specimen). By N. Dalton, M.D. April 17, 1888.

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15. Advanced surgical kidneys. By T. F. Pearse, M.D. March 20, 1888.
16. Epithelioma of the kidney associated with calculus. By Sir W. Mac Cormac. March 6, 1888.
17. Bladder with vesical and prostatic calculi. By C. Mansell Moullin. May 1, 1888.
18. Villous tumour of bladder; surgical kidney. By C. Heath. October 18, 1887.

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19. Transverse fracture of patella united by bone. By C. Mansell Moullin. February 7, 1888.
20. Syphilitic crania. By C. Mansell Moullin. March 6, 1888.
21. Tumour of bone. By C. Stonham. February 21, 1888.
    (1) Spindle-celled sarcoma of humerus with fracture of the shaft of the bone (University College Museum, 664[A]).
    (2) Spindle-celled sarcoma of the tibia (University College Museum, 661[A]).
    (3) Cystic myeloid sarcoma of the head of the tibia, involving the joint and the lower epiphysis of the femur.
    (4) Cystic sarcoma of the femur.
    (5) Spindle-celled sarcoma of the femur.
22. Tabetic feet. By J. R. Lunn. May 15, 1888.

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23. Osteoma of the skin. By C. Mansell Moullin. May 1, 1888.
24. Dermoid cyst from the thyrohyoid space (third branchial cleft). Two specimens from different cases. By S. G. Shattock. February 21, 1888.

25. Leprosy and diffuse frambœsia of the hands. By C. Stonham, for Beaven Rake, M.D. (Trinidad). Specimen preserved in University College Museum. February 7, 1888.
26. Gangrene of the leg following typhoid fever (recent specimen). By F. D. Drewitt, M.D. December 20, 1887.
27. Photographs of a case of atrophy of the arm with œdema and ulceration of the hand. By E. Jacob, M.D. October 18, 1887.

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